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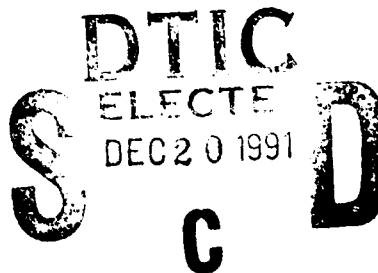
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Evaluation of Critical Care Nurses'

Knowledge and Ability to Utilize Information
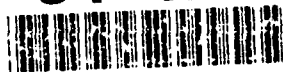
Related to Pulmonary Artery Pressure Measurement

by

Elizabeth J Bridges

A thesis submitted in partial fulfillment
of the requirements for the degree

Master of Nursing

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Approved by *Susan L. Woods*
(Chairperson of Supervisory Committee)

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University of Washington

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Information Related to Pulmonary Artery Pressure Measurement

by Elizabeth J. Bridges

Chairperson of Supervisory Committee:

Associate Professor Susan L. Woods,

School of Nursing

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ACKNOWLEDGEMENTS

CHAPTER I

Problem Statement

To assist in the therapeutic modification of the critically ill patient's response to various disease states, the critical care nurse must possess a high level of knowledge of how disease processes alter specific physiologic parameters as well as the techniques used to monitor these parameters. Straw (1986) evaluated critical care nurses' knowledge of pulmonary artery (PA) pressure measurement and reported that scores on a criterion-based evaluation of this knowledge ranged from 20% to 90%, with a mean of 59.2%. Only 23.7% of the nurses scored higher than 70% on the test. In 1987, Dolter studied critical care nurses' knowledge of physiological, pathophysiological, and technical aspects of PA wedge pressure measurement, external and internal artifact effects on PA wedge pressure measurement, and complications of PA monitoring. The average score on this test was 30.8%.

The importance of this apparent knowledge deficit is magnified by research conducted by Holmes (1982). Holmes' study reported that 61% of all pediatric and adult critical care units utilize PA catheters for monitoring. Ninety percent of all adult critical care units reported using PA catheters. In addition, in 57% of all units, nurses were responsible for the set up of the PA catheter system. In 59% of the facilities, nurses were responsible for reading PA catheter data, calibrating the catheter system, and collecting PA catheter blood samples. In addition, in 47% of the facilities,

nurses were responsible for acting on the data collected (the study did not specify what this latter activity entailed). In order to accomplish these tasks, there is clearly a need for nurses in these critical care units to have knowledge of PA pressure measurement as well as the ability to interpret and utilize the collected data.

The PA catheter is a complex piece of equipment. The safe, effective use of the PA catheter and utilization of the hemodynamic data provided requires an extensive knowledge base. Utilization of the data provided by the PA monitoring system requires knowledge related to the mechanics of operation, the interrelated physiologic parameters monitored, and the alteration of these physiologic parameters in various disease states.

If a critical care nurse has a knowledge deficit related to recognition of pathognomonic alterations in hemodynamic parameters, or complications associated with PA pressure measurement, therapeutic mismanagement or potentially severe injury to the patient may occur. In addition, lack of knowledge may lead to incorrect technique related to positioning the patient prior to pressure measurement, evaluating the technical aspects of the equipment, interpretating the pressure waveforms, or wedging the balloon. This incorrect technique will further increase the risk for therapeutic mismanagement or injury to the patient.

With respect to professional responsibility, the American Association of Critical-Care Nurses (AACN) Standards for Nursing Care of the Critically Ill (Sanford & Disch, 1989) serves as the basis for

the practice of critical care nursing. This document stated that the critical care nurse must "demonstrate technical competency in gathering objective data" (p. 6), "integrate current scientific knowledge with technical and psychomotor competence" (p. 11), "compare patient's response with expected results" and "base the evaluation on data from pertinent sources." (p. 13). In addition, the nurse must "attempt to determine the cause of any significant difference between the patient's response and the expected response" (p. 13). The ability to meet any of these standards would be severely constrained by the lack of knowledge reported in both Straw's (1986) and Dolter's (1987) studies of critical care nurses.

In addition to the AACN Standards, Disch (1980) stated critical care nurses must participate in "ongoing educational activities...to acquire advanced knowledge of psychosocial, physiological, and therapeutic components specific to the care of the critically ill" (p. 19). This point is reiterated in the AACN position statement on the "Scope of Critical Care Nursing Practice" (1986) that indicated critical care nurses must respond to demands placed on them for the use of advanced knowledge through ongoing education. The level of education is based on the nurses' expanding knowledge and experience level.

Straw's (1986) and Dolter's (1987) studies were the only studies found that evaluated critical care nurses' knowledge of PA pressure measurement. While both studies identified a global knowledge deficit, they did not identify specific learning needs. No

data-based study evaluating critical care nurses' ability to utilize knowledge of PA pressure measurement in a patient care situation was found during a review of the literature. It is important to assess and identify the learning needs of critical care nurses, with respect to their knowledge of PA pressure measurement, as well as their ability to utilize this knowledge. This research project, which sought to assess and identify specific learning needs, represented the first step in the design of an instructional intervention to improve critical care nurses' knowledge and ability to utilize information related to PA pressure measurement in a clinical situation.

This study expanded upon the work of Straw (1986) and Dolter (1987) with respect to critical care nurses' knowledge of PA pressure measurement. The goal of this study was to describe critical care nurses' knowledge of PA pressure measurement, and their ability to utilize this information in the care of a patient requiring PA pressure monitoring.

CHAPTER II

Conceptual Framework

Pulmonary artery (PA) pressure measurement is a skill that requires a broad knowledge base. Difficulty arises when one tries to evaluate this complex knowledge base in a consistent manner. This chapter contains a discussion of the use of a cognitive taxonomy to describe expected clinical behaviors, use of written simulation as a method to evaluate critical care nurses' knowledge base related to PA pressure measurement, and the rationale for use of a criterion-referenced instrument to assess this knowledge base. A summary and purpose statement conclude this chapter.

Pulmonary Artery Pressure Measurement

The PA catheter is one component of a complex monitoring device that facilitates the management of critically ill patients. The keys to successful use of the PA catheter and PA pressure measurement include the critical care nurses knowledge and ability to utilize information related to the physiologic parameters indirectly measured by the PA catheter, e.g., preload, afterload, and contractility, and an understanding of how these parameters are altered by various pathophysiologic processes; and the technical variables that can affect the accuracy of PA pressure measurement, e.g., pulmonary effects, patient positioning, waveform interpretation, reference level, PA pressure stabilization, and normal PA pressure stabilization. In addition, the nurse must possess the ability to troubleshoot the monitoring equipment and evaluate the patient for

potential complications associated with the presence of the
indwelling PA catheter, such as arrhythmias, infection, and pulmonary
infarction or perforation. Finally, the critical care nurse must be
able to utilize this knowledge and information in specific patient
situations. As noted in the study by Holmes (1982), the critical
care nurse is responsible for a large portion of these duties. Both
Straw (1986) and Dolter (1987) have attempted to evaluate this
knowledge base. In addition to the evaluation of nurses' knowledge
in this area, the evaluation of the how nurses utilize this knowledge
in specific clinical situations is essential.

Physiology of Pulmonary Artery Pressure Monitoring

Cardiac output, the amount of blood ejected from the ventricle
each minute, is one of the primary determinants of oxygen delivery to
the tissues. Cardiac output is the product of stroke volume and
heart rate. Five factors are known to affect stroke volume: 1)
preload, 2) afterload, 3) contractility, 4) contractile synergy, and
5) heart rate. This section addresses each of these factors with
regard to their physiologic definitions and the basis for their
measurement.

Preload.

Preload refers to end-diastolic myocardial fiber length, which
in the intact ventricle is related to the end-diastolic volume (Shah,
1983). The importance of fiber length is explained by Starling's law
of the heart (1914) that stated within given limits, an increase in
fiber length is associated with an increase in force of contraction.

The increased force of contraction results in an increased stroke volume, the amount of blood ejected from the heart with each beat. This length-tension relationship allows for equalization of right and left heart output. For example, if right ventricular volume is increased, the fiber length of the right ventricle is increased, and the force of the subsequent contraction is increased. The increased contractile force results in an increase in stroke volume to the left ventricle. The left ventricular fibers are then stretched, with a subsequent increase in force of contraction and stroke volume (Bond, 1989).

In the intact ventricle, measurement of fiber length is difficult; therefore, end-diastolic volume is used to estimate resting muscle length (Hedges, 1983). However, use of end-diastolic volume assumes there is uniform distention of all fibers in the ventricle as the volume is increased, which is not the case (Van Aken & Vendermeersch, 1988). It is technically difficult to measure end-diastolic volume in a clinical setting; therefore, measurement of end-diastolic pressure is used. Use of end-diastolic pressure assumes a fixed relationship between pressure and volume. As can be seen in Figure 1, the pressure-volume relationship of the ventricle is curvilinear. In a normal ventricle (curve I) at lower end-diastolic volumes, a large change in volume is accompanied by a small change in pressure (point A), but as the end-diastolic volume increases, volume changes are accompanied by larger changes in end-
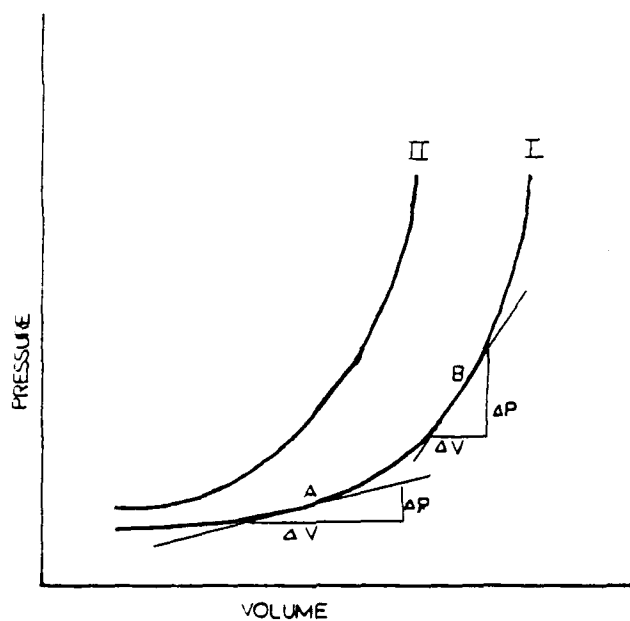
Figure 1. Typical pressure-volume curve. Curve I reflects a normal ventricle; Curve II represents a ventricle with decreased compliance. Points A and B reflect the effect of volume on ventricular compliance. P = pressure; V = volume; Δ= change

diastolic pressure (point B). This pressure-volume relationship is a reflection of ventricular compliance. Compliance (the inverse of stiffness) refers to ventricular distensibility, and is defined by the slope of the pressure-volume curve, or the change in pressure for any given change in volume. Any alteration in compliance will affect the pressure-volume relationship, and limit the usefulness of end-diastolic pressure as an indicator of fiber length. For example, in myocardial ischemia, compliance is decreased (curve II), i.e., any given change in volume is accompanied by a larger change in pressure when compared with a normal curve (curve I). Therefore, the relationship between end-diastolic volume and end-diastolic pressure is not constant, and may be affected by the diastolic compliance of the ventricle (curve I vs curve II) or the end-diastolic volume (point A vs Point B) or both (Sibbald, Calvin, & Driedger, 1982). Caution; therefore, must be used when approximating end-diastolic volume with end-diastolic pressure.

Afterload.

Afterload is the wall tension that is developed during ventricular systole, and is related to the force opposing ventricular ejection (Hurst, 1984). An alteration in afterload inversely affects the velocity of myocardial shortening, i.e., an increase in afterload is associated with a decrease in velocity. Clinically, systemic vascular resistance (SVR) is used as the indicator of left ventricular afterload (Bond, 1989). In the normal ventricle, an increase in afterload (increased impedance to ejection) results in

little change in stroke volume; however, in the presence of
myocardial dysfunction, an increase in afterload may result in a
dramatic reduction in stroke volume (Shah, 1983). Afterload can be
clinically modified by the use of vasodilator or vasoconstrictor
therapy.

Also of importance is the effect of afterload on myocardial
oxygen consumption. One of the primary determinants of myocardial
oxygen consumption is the development of tension during isovolumetric
contraction. An increase in afterload necessitates an increase in
ventricular tension and the subsequent increase in myocardial oxygen
consumption (Bond, 1989).

Contractility.

Contractility is defined as the maximum velocity of cardiac
muscle shortening, independent of changes in preload and afterload.
An increase in stroke volume without a change in preload or afterload
is the result of increased contractility. Factors that increase
contracility are known as inotropes, and include catecholamines,
dopamine, dobutamine, calcium, and digoxin. Negative inotropic
agents and clinical situations,that decrease contractility, include
beta blockers, hypoxia, acidosis, myocardial ischemia and infarction,
barbiturates, procainamide, and quinidine (Shah, 1983; Bond, 1989).

Synergy of contraction.

Stroke volume is also affected by the synergy of ventricular
wall motion. Direct assessment of contractile synergy is made
through the use of contrast angiography, while measurement of the

ejection fraction provides an indirect index of ventricular function.
In the presence of myocardial injury, four abnormal contraction
patterns (asynergy) occur: 1) asynchrony, a dissociation in the
contraction of adjacent wall segments, 2) asyneresis or hypokinesis,
a localized reduction in myocardial contraction, 3) akinesis, an
absence of ventricular wall motion, and 4) dyskinesis, paradoxical
wall movement during systole, i.e., bulging. The contractile
asynergy causes a decrease in left ventricular stroke volume, and
potentiates heart failure (Glasser, 1977). However, the decrease in
stroke volume may be offset by hyperkinesis of the unininvolved
ventricular areas (Herman, Heile, Klein, & Gorlin, 1967; Pasternak,
Braunwald & Sobel, 1988; Newton, 1989). The hyperkinetic
compensatory action of the uninvolved areas limits the efficacy of
ejection fraction determinations, which may reflect a normal global
ventricular function despite contractile asynchrony (Braunwald,
1988).

Contractile abnormalities that involve 10% to 20% of the left
ventricle do not usually affect the ejection fraction or end-
diastolic volume. When contractile abnormalities involve 25% to 30%
of the LV mass, there is usually an increase in end-diastolic volume
and a concomitant decrease in ejection fraction (Kennedy, 1976;
Glasser, 1977; Newton, 1989). Generally congestive heart failure
does not occur until the ejection fraction is decreased below 30%
(normal = 67%) (Kennedy, 1976). Myocardial injury, and the resultant
contractile abnormalities, involving greater than 40% of the

ventricular wall mass is associated with the development of cardiogenic shock (Swan, Forrester, Diamond, Chatterjee, & Parmley, 1972; Waller, 1988).

Heart rate.

An increase in heart rate is associated with an increased influx of calcium into the sarcoplasmic reticulum (Treppe effect), and a subsequent increase in the force of contraction and stroke volume (Bond, 1989; Huntsman & Feigl, 1989). However, the effect of the increased force of contraction on stroke volume may be offset by a decrease in diastolic filling time and coronary artery perfusion.

The Pulmonary Artery Catheter

The flow-directed, balloon-tipped pulmonary artery (PA) catheter was introduced in 1970 by Swan and coworkers (Swan, Ganz, Forrester, Marcus, Diamond, & Chonette, 1970). Today there are a large number of PA catheters produced, but all are essentially similar in structure and function. The PA catheter (Figure 2) is a triple or quadruple lumen, polyvinylchloride catheter with a variable external diameter. The catheter is percutaneously inserted through the internal jugular, external jugular, subclavian, femoral, or antecubital vein (Matthay, Wiedemann, & Matthay, 1985). Depending on the site of insertion, the PA catheter tip will enter the superior vena cava and right atrium at different distances, denoted by the incremental markings every 10 cm on the catheter. Once the right atrium is reached the balloon is inflated and the catheter is
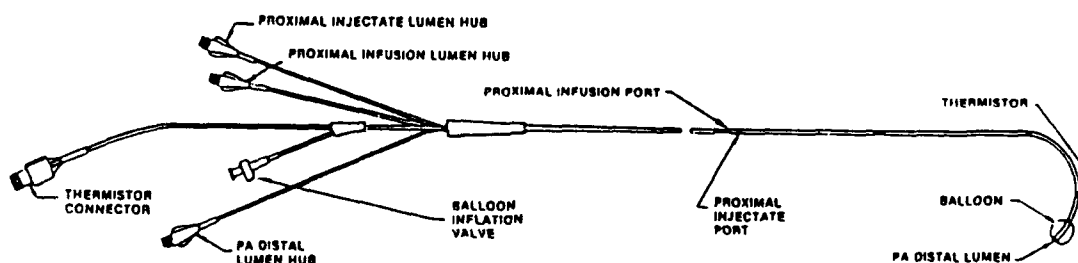
Lumen 1: The distal lumen terminates at the tip of the catheter. Chamber pressures, PA pressure, and PA wedge pressure, as well as blood samples, can be obtained through this lumen.

Lumen 2: The proximal lumen terminates 30 cm from the catheter tip, placing it in the right atrium when the distal lumen opening is in the pulmonary artery. Lumen 2 carries the injectate necessary for cardiac output computation and may also be used for infusion of solutions. By connecting the proper pressure transducer to the lumen, right atrial pressure can be monitored.

Lumen 3: This lumen contains the electrical leads for the thermistor, which is positioned on the catheter surface 4 cm proximal to its tip.

Lumen 4: This lumen is used to inflate and deflate the 1.5 ml capacity balloon.

Balloon: The inflated balloon serves two purposes in the insertion procedure. First, it assists in pulling the catheter through the chambers of the heart due to fluid dynamic drag on the balloon. Second, the fully inflated balloon covers the catheter tip; this distributes the tip force over a larger area and reduces the occurrence of premature ventricular contraction caused by catheter tip irritation to the endocardium.

Figure 2. The Swan-Ganz[R] heparin coated flow-directed thermodilution catheter. From "The Swan-Ganz[R] Venous Infusion Port (VIP[TM]) Thermodilution Catheter" Baxter Healthcare Corporation, Santa Ana, CA. Copyright Baxter Healthcare Corporation. Reprinted with permission.

"floated" through the right atrium and right ventricle and out into the PA where it wedges (Swan, 1975; Wiedemann, Matthay, & Wiedemann, 1984). Once a characteristic pulmonary artery wedge pressure tracing has been attained, the balloon is deflated, allowing the catheter to recoil slightly into the PA. The balloon is again slowly inflated under continous monitoring of the PA pressure tracing until the waveform changes from a PA to a PA wedge pressure tracing. The volume of air required to inflate the syringe should be noted, and the balloon again deflated. Following deflation, the monitored waveform should return to a PA configuration (Civetta & Gabel, 1972; Swan & Ganz, 1975). The position of the catheter is determined by the characteristic waveforms from each chamber, and the pulmonary artery.

Waveform characteristics

Four pressure waveforms are evaluated clinically to differentiate varying disease pathologies. Accurate interpretation of each waveform is important. This section describes the characteristic waveforms, methods for interpretation, and the factors that may alter the waveform characteristics and the pressures that are measured.

Right atrial pressure.

The right atrial (RA) pressure or central venous pressure (CVP) consists of three positive deflections (Figure 3A). The "a" wave is due to atrial contraction, and follows the P wave on the electrocardiogram (ECG) tracing. The "a" wave is followed by the "x"
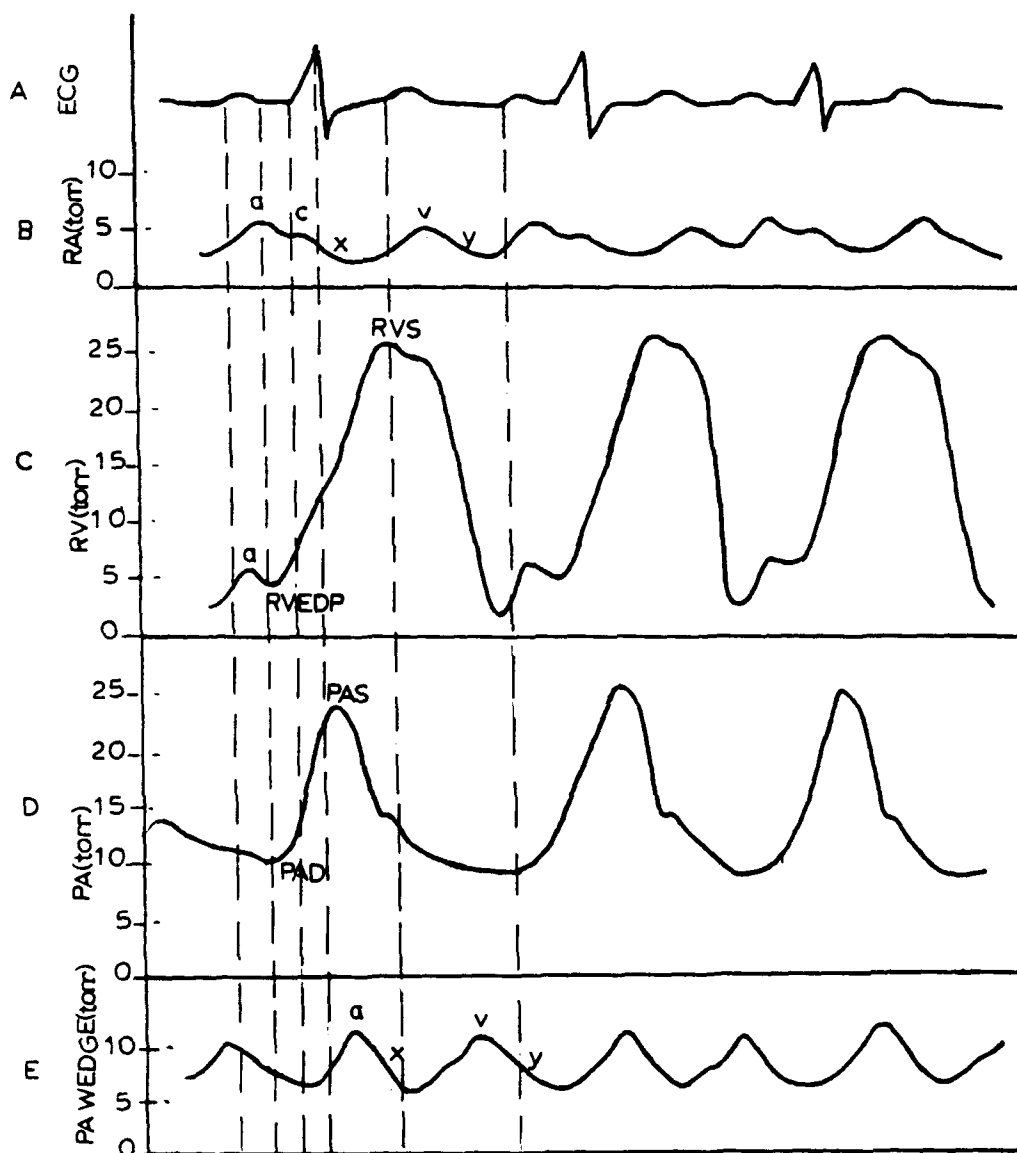
Figure 3. Pressure waveforms recorded by the pulmonary artery
catheter: A) electrocardiogram (ECG) tracing; B) right atrial
pressure (RAP); C) right ventricular (RV) pressure; D) pulmonary
artery pressure (PA); E) pulmonary artery wedge (PA wedge) pressure.
RVS = right ventricular systolic, RVD = right ventricular diastolic,
PAS = pulmonary artery systolic, PAD = pulmonary artery diastolic

descent, which is related to atrial relaxation. On the downslope of the "x" descent, the "c" wave may occur. The "c" wave is due to motion of the tricuspid valve towards the atria, at the onset of right ventricular systole, and occurs during the P-R interval of the ECG. The distance between the "a" and "c" wave is the same distance as the P-R interval on the ECG. After the nadir of the "x" descent, the pressure in the atrium begins to rise as a result of venous filling, and the pressure peaks just before opening of the tricuspid valve. The "v" wave occurs near the end of the T wave on the ECG. Following the "v" wave, the right atrium empties into the ventricle and the pressure in the atrium decreases ("y" descent) (Bertrand & Widimsky, 1985; Sharkey, 1987; Braunwald, 1988). The RA pressure is measured as a mean, with a normal value of 0 to 8 torr (1 torr = 1 mm Hg).

The RA pressure is an indirect indicator of right ventricular filling pressure (Swan, 1975). An RA pressure less than 2 torr may indicate hypovolemia, vasodilatation, or increased myocardial contractility, while a RA pressure greater than 8 torr may be associated with hypervolemia, vasoconstriction, decreased myocardial contractility, right ventricular failure, tricuspid insufficiency, positive pressure ventilation, pericardial tamponade, pulmonary embolus, and obstructive pulmonary disease (Gardner & Woods, 1989). A more specific analysis of the RA waveform may prove diagnostic. An elevated "a" wave may be associated with right ventricular failure

(decreased compliance), or tricuspid stenosis. An elevated "v" wave may be associated with tricuspid insufficiency (Darovic, 1987).

Use of the RA pressure to extrapolate information regarding the left side of the heart must be done with caution. Samii, Conseiller, and Viars (1976) studied 13 patients without cardiac or pulmonary disease and found that RA pressure was strongly correlated with PA wedge pressure (r = 0.71, p < 0.001); however, there was a wide range of PA wedge pressures for any given RA pressure. In addition, in patients with a RA pressure greater than 7.5 torr (10 cm H_2O), RA pressure was significantly, but poorly correlated with PA wedge pressure (r = 0.22, p < 0.05). In patients with acute myocardial infarction, RA pressure was poorly correlated with PA wedge pressure (r = 0.45) (Forrester, Diamond, Mchugh, & Swan, 1971). In addition, in the latter study, RA pressure was not related to the presence or absence of radiologic abnormalities consistent with pulmonary vascular congestion. It was therefore recommended that RA pressure not be used as a criterion for determination of left ventricular (LV) dysfunction, or to guide fluid therapy in patients with known LV dysfunction (Forrester, Chatterjee, & Swan, 1973).

Right ventricular pressure.

Right ventricular (RV) pressure is not continuously monitored. However, recognition of the RV waveform is important during flotation of the catheter and following correct positioning of the PA catheter. The presence of a RV waveform, following correct positioning of the

catheter, indicates that the PA catheter has slipped back into the right ventricle.

The RV pressure waveform (Figure 3B) consists of an early rapid filling phase (60% of ventricular filling), a slow filling phase (25% of ventricular filling), and an atrial systolic wave ("a" wave) that is responsible for approximately 15% of filling. Because of the low resistance to flow across the tricuspid valve during diastole, the RV diastolic pressure is approximately equal to RA pressure. Right ventricular end-diastolic pressure is measured immediately after the "a" wave (Braunwald, 1988), with a normal range of 0 to 8 torr. Right ventricular systolic pressure, is measured at the peak of the pressure wave, with a normal range of 15 to 30 torr.

Factors that may increase RV systolic pressure include: obstructive lung disease, pulmonary embolism, hypoxemia, adult respiratory distress syndrome, and pulmonary vascular overload due to left ventricular dysfunction or left-to-right shunts. Right ventricular diastolic pressure is affected by all the factors that increase RA pressure. A decrease in RV diastolic may be due to hypovolemia, and a decrease in RV systolic may be associated with RV failure (Darovic, 1987).

Pulmonary artery pressure.

Three pulmonary artery pressures are measured: systolic, diastolic, and mean (Figure 3C). The systolic upstroke is due to blood flow from the right ventricle into the PA; therefore, PA systolic is normally equal to RV systolic pressure (15 to 30 torr).

As RV ejection ends, PA pressure drops. At the point where RV pressure is less than PA pressure, the tricuspid valve closes, resulting in an incisura (dicrotic notch) on the descending waveform. Diastolic pressure in the PA is higher than RV diastolic pressure because of the closure of the tricuspid valve (this point is useful in differentiating the two waveforms). The PA waveform corresponds with the QRS complex of the ECG. End-diastolic pressure is measured immediately before systole (Braunwald, 1988; Darovic, 1987), and normally ranges from 3 to 12 torr. Mean PA pressure normally ranges from 9 to 16 torr, and as demonstrated in Figure 4A is measured by bisecting the end-expiratory pressure waveform so that there are equal areas above and below the bisection (Gardner & Woods, 1989). In the presence of normal pulmonary vasculature, and the absence of left heart venous obstruction (left atrial myxoma or mitral stenosis), pulmonary artery end-diastolic pressure (PAEDP) is approximately equal to LV pressure. As noted in Figure 5A, during diastole, when the mitral valve is open, an unobstructed column of blood exists from the PA to the left atrium and the left ventricle. Therefore, pressure is approximately equal across all three areas (Gardner & Woods, 1989). As noted above, knowledge of LV end-diastolic pressure (LVEDP) provides a guide for therapeutic modification of the five factors that affect cardiac output.

Factors that can increase PA pressures include left heart dysfunction, mitral stenosis and insufficiency, LV failure, intravascular volume overload, decreased LV compliance, and

A.

B.

Inspiration   Expiration
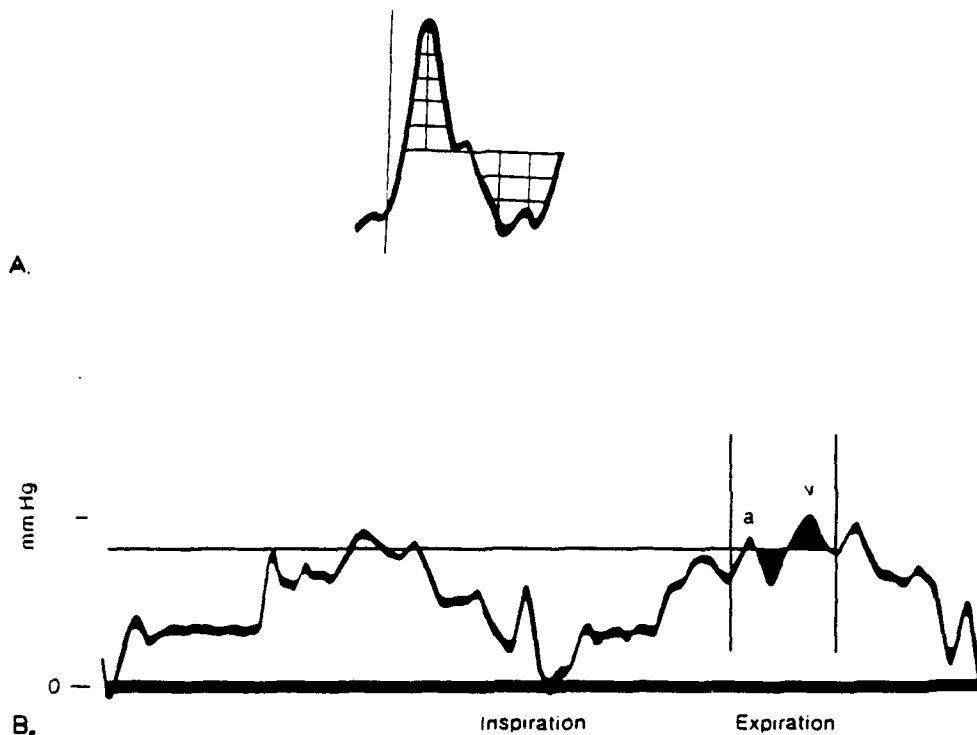
Figure 4A. The pulmonary artery mean is measured by bisecting the
end-expiratory pressure waveform so that there is equal area above
and below the bisection. Figure 4B. The PA wedge pressure is
measured by bisecting the "a" and "v" wave so there is equal area
above and below the bisection. Measurements must be obtained using
the portion of the waveform at end-expiration. From Cardiac Nursing
(p. 462) by S.L. Underhill. S.L. Woods, E.S. Sivarjan Froelicher, &
C.J. Halpenny (Eds.), 1989, Philadelphia: J.B. Lippincott.
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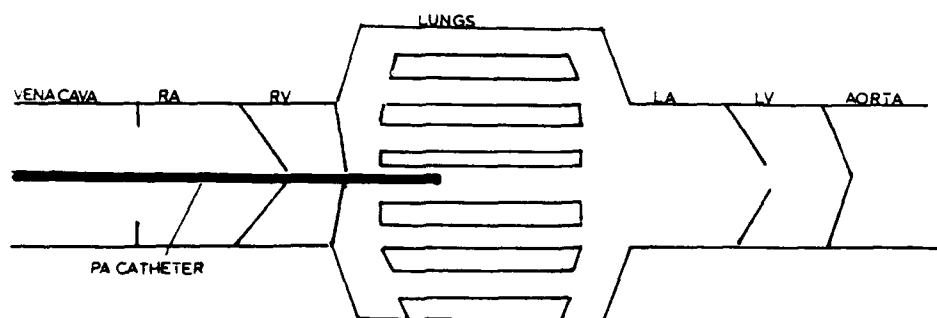
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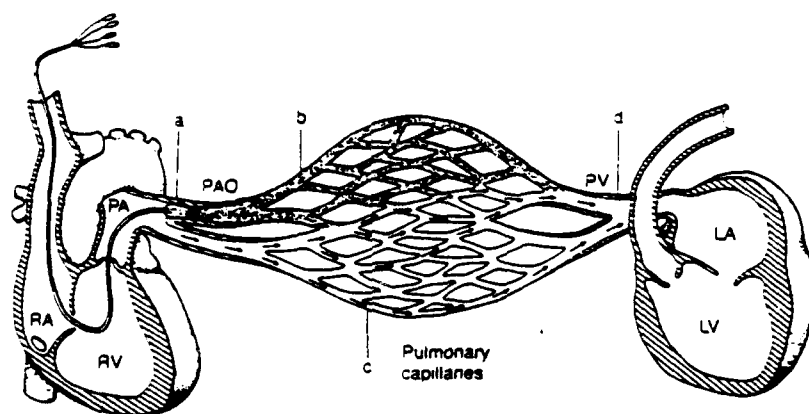
B.

Figure 5A. Schematic of pulmonary artery catheter in the heart at end-diastole. Pressures measured by the distal port (PA pressure) reflect end-diastolic pressures. RA = right atrium; RV = right ventricle, LA = left atrium; LV = left ventricle.

Figure 5B. Schematic representation of the PA catheter in the wedge position. From its position in a small occluded segment of the pulmonary circulation, the wedged PA catheter allows the electronic monitoring equipment to "look through" a non-active segment of the pulmonary circulation to the hemodynamically active pulmonary veins and left atrium. From Hemodynamic Monitoring (p. 149) by G. Darovic, 1987. Philadelphia: W.B. Saunders. Copyright 1987 by W.B. Saunders. Reprinted by permission.

pericardial tamponade or effusion. A decrease in PA pressures may be associated with hypovolemia.

Pulmonary artery wedge pressure.

The PA wedge pressure is determined by inflation of the balloon on the distal end of the PA catheter, which allows the catheter to float forward to wedge in a segment of the PA. The obstruction of blood flow creates a non-moving column of blood (Figure 5B) that allows transmission of the LA pressure wave. The pressure-sensor, located on the distal end of the catheter, records the pressure from the point where the non-moving blood meets moving blood (from non-occluded segments of the PA). At end-diastole, in the presence of an open column of blood between the pulmonary vasculature and left atrium and ventricle, the recorded occlusion pressure will reflect LA and LV pressures (Darovic, 1987).

The PA wedge pressure waveform is similar to LA pressure, but is slightly damped, and phase delayed due to pulmonary vascular transmission (Swan, 1975). The PA wedge pressure waveform consists of two positive deflections. The "a" wave, which reflects LA systole, follows the P-R interval on the ECG (slightly later than the RA "a" wave, due to delayed transmission) (Sharkey, 1987). The "v" wave reflects LV systole, and LA filling. The peak of the "v" wave occurs during the T-P interval on the ECG. The PA wedge pressure is measured as a mean pressure. As noted in Figure 4B, the pressure is measured by bisecting the "a" and "v" waves so there is an equal area above and below the bisection. The measurements are made on the

portion of the waveform that represents end-expiration (Gardner & Woods, 1989).

Factors that may increase the PA wedge pressure include intravascular volume overload, left heart dysfunction or failure, mitral stenosis or insufficiency, decreased LV compliance, and pericardial tamponade or effusion. Hypovolemia may be associated with a decrease in PA wedge pressure (Darovic, 1987).

Use of the PA Catheter in Patients with Myocardial Infarction

Coronary heart disease affects approximately five million Americans. Myocardial infarction (MI) caused 513,700 deaths in 1987, and is the leading cause of death in America today (American Heart Association [AHA], 1989). A population-based study of 3,263 patients hospitalized for acute MI was conducted to determine the association of PA catheter use with in-hospital prognosis and length of hospital stay (Gore, Goldberg, Spodick, Alpert, & Dalen, 1987). The use of the PA catheter to guide therapy in these patients was associated with an increased length of hospital stay, and increased mortality in patients with hypotension or congestive heart failure. Use of the PA catheter was associated with a slightly lower fatality rate in patients with cardiogenic shock. Because of the high prevalence of MIs in the population, and the questionable benefit of PA catheter pressure measurement to patients suffering a MI, this section discusses use of the PA catheter in patients with a MI, factors that may decrease the accuracy of PA pressure measurement as an index of LV function, and how data derived from the PA catheter may be used to

more accurately assess and guide therapeutic measures in the patient
with a MI.

Pulmonary artery-left ventricular pressure relationship.

As noted, the PAEDP and PA wedge pressure are used as indices
of LVEDP and LV end-diastolic volume. A large number of research
studies have been conducted to validate this relationship (Appendix
A). In patients with normal LV function, mean LA pressure (Braunwald
& Brockenbrough, 1961) and PA end-diastolic (Bouchard, Gault, & Ross,
1971) pressures were found to be good indicators of LV end-diastolic
pressure. However, studies in patients with LV dysfunction are less
conclusive (Appendix A). In patients suffering from an acute MI or
LV dysfunction, PAEDP and PA wedge pressure failed to consistently
reflect changes in left heart pressures (Falicov & Resnekov, 1970;
Forrester, Diamond, Ganz, Swan, & Danzig, 1970; Bouchard & et al.,
1971; Rahimtoola et al., 1972; Fisher, DeFelice, & Parisi, 1975;
Calvin, Driedger, & Sibbald, 1981).

In patients with LV dysfunction, Braunwald and coworkers (1961)
found that LA pressure was a good indicator of LVEDP. In addition,
PAEDP and PA wedge pressure were found to be good indicators of left
heart pressures (Kaltman, Herbert, Conroy, & Kossman, 1966; Jenkins,
Bradley & Branthwaite, 1970; Forsberg, 1971; Scheinman, Evans, Weiss,
& Rapaport, 1973; Walston & Kendall, 1973). Lipp-Ziff and Kawanishi
(1991) studied 100 cardiac patients to determine the most accurate
measurement of LVEDP based on PA diastolic pressure relative to the
QRS complex. Pulmonary artery diastolic pressure was measured at

three intervals relative to the QRS complex: 1) PA diastolic at the lowest point on the waveform, 2) PA diastolic 0.04 second after the onset of the QRS complex, and 3) PA diastolic 0.08 second after the onset of the QRS complex. Lipp-Ziff and Kawanishi found that in patients with cardiac disease, in normal sinus rhythm and pulmonary vascular resistance, the PA diastolic pressure measured 0.08 second after the onset of the QRS complex had the best correlation ($r = 0.88$) with LVEDP. However, the difference between all three PA diastolic pressure measurements was not clinically significant.

In the presence of LV dysfunction, measurement of the PA "a" wave pressure was found to provide a more precise measurement of LV diastolic pressure (Rahimtoola et al., 1972), and LVEDP, even when PA and PA wedge pressures failed to reflect LVEDP (Falicov & Resnekov, 1970; Bouchard et al., 1971; Balcon, Bennett, & Sowton, 1972; Scheinman et al., 1973; Fisher et al., 1975). The increased accuracy of the "a" wave pressure was more frequently observed in patients with decreased LV compliance and reflected atrial contraction into a stiff ventricle. In such cases, the "a" wave peak was found to provide a more reliable index of LVEDP than PA wedge pressure, which failed to reflect the absolute height of the "a" wave (Rahimtoola et al., 1972; Swan, 1975; Raper & Sibbald, 1987). However, in all of these studies clinically important individual variations occurred.

Saadjian, Cassot, and Torresani (1981) also found that the location of the MI affects these pressure relationships. Inferior/posterior infarcts ($n = 13$) were associated with a higher

degree of RV dysfunction. This RV dysfunction was associated with a decreased correlation between PAEDP and LVEDP (r = 0.75, p < 0.001) when compared with inferior (n = 31; r = 0.90, p < 0.001), anteroseptal (n = 25; r = 0.93, p < 0.001), and anterolateral (n = 40; r = 0.88, p < 0.001) infarcts. Therefore, use of PA pressures as indices of left heart pressures in the presence of myocardial infarction or LV dysfunction must be viewed with caution.

Factors that affect the pulmonary artery-left ventricular

pressure relationship in patients with a myocardial infarction.

The underlying pathology of an MI introduces several factors that may explain the discrepancy between PA and left heart pressure measurements. Such factors include an elevation in pulmonary vascular resistance (PVR) secondary to hypoxic vasoconstriction, an increase in heart rate in an attempt to increase cardiac output, and altered myocardial compliance (Gorlin, 1977). In patients with normal or slightly elevated PVR, Jenkins and coworkers (1970) found a high correlation between mean LA pressure and PAEDP (r = 0.95 and r = 0.87 respectively). These findings were similar to those of Forsberg (1971), and Rao and Sissman (1971). However, in 20 children with congenital heart disease and an elevated PVR, Rao and Sissman ((1971) found a wide and inconsistent relationship between the PA venous wedge pressure and all PA pressures. In addition, Jenkins and coworkers (1970) found large differences between LA pressure and PAEDP. On the contrary, Levin and Glassman (1985), in a retrospective review of 10,000 cardiac catheterizations in patients

with chronically elevated PVR due to valvular or ischemic heart disease, found a significant correlation ($r = 0.84$, $p < 0.001$) between PA wedge pressure and LA pressure regardless of the level of PVR. Limitations of the latter study, however, include its retrospective nature and the use of patients with chronically elevated PVR. Therefore, in patients with an acute elevation in PVR, use of PA diastolic pressure as an index of PA wedge or LA pressures was not recommended.

An elevation in heart rate above 115 to 130 beats per minute has been found to limit the efficacy of PAEDP as an index of LVEDP (Bouchard et al., 1971, Yenson, Wood, Mantaras, & Harvey, 1977). In a study of 15 dogs with complete atrioventricular dissociation, Yenson and coworkers (1977) noted that heart rate explained 58% of the variation in PAEDP and LVEDP, independent of changes in cardiac output. Of note, in both of these studies is that the disparity in pressures was related to a decrease in LVEDP and an increase in PAEDP. The heart rate induced change in the relationship of these two pressures was thought to be related to inadequate time for equilibration of the pressures at end-diastole. Both of the studies were limited by the use of subjects with normal cardiac function. In a study of 15 patients (seven with abnormal LV function), McLaurin, Rolett, and Grossman (1973) confirmed that tachycardia does cause a decrease in LVEDP in patients with normal ventricular function. However, in patients with LV dysfunction, LVEDP increased as a result of pacing induced tachycardia. In the latter group of patients, the

increase in LVEDP was associated with signs and symptoms of ischemia, and depressed myocardial relaxation. Based on these studies, caution should be taken when using PAEDP as an index of LVEDP in patients with heart rates greater than 115. Further research is needed to clarify the effect of heart rate on the PAEDP-LVEDP relationship in patients with LV dysfunction.

The third factor affecting the PA-LV pressure relationship is an alteration in myocardial compliance (Figure 1). In the presence of myocardial disease, the pressure-volume relationship may be altered, due a change in myocardial compliance. If the ventricle becomes less compliant because of fibrosis or hypertrophy, a large increase in filling pressure may occur with only a small increase in volume (Gorlin, 1977). Clinically, while absolute end-diastolic volume changes cannot be measured, certain assumptions about compliance can be made. If a large change in PA wedge pressure occurs as a result of a small volume challenge, one can make the assumption that the ventricle is noncompliant; and further volume replacement may increase PA wedge pressure to a point where pulmonary edema develops (Sibbald et al., 1982).

While the PA wedge pressure may be a less than optimal indicator of LV volume in the MI patient, it is an accurate index of the propensity for the development of pulmonary edema. The relationship between PA wedge pressure and microvascular fluid flux

can be explained by the Starling (1896) equation:

Fluid flux = $Kf \, (P_{cap} - P_i) - f(\pi_{cap} - \pi_i)$

Kf = capillary membrane permeability;

P_{cap} = capillary hydrostatic pressure;

P_i = interstitial hydrostatic pressure;

σf = solubility coefficient (ease with which substance passes through the membrane);

π_{cap} = capillary oncotic pressure;

π_i = interstitial oncotic pressure.

Basically the Starling equation indicates that P_{cap} and π_i favor fluid movement out of the capillary into the interstitial spaces, while P_i and π_{cap} oppose this fluid flux. In the lung there is normally a net fluid flux out of the pulmonary capillaries into the interstitium, where the fluid is removed by the lymphatic system.

Pulmonary artery wedge pressure is an indirect indicator of pulmonary capillary hydrostatic pressure. Therefore, any increase in PA wedge pressure may result in an increase in the net flux of fluid out of capillary vessels, into the pulmonary interstitial space. If the lymphatic system fails to remove the excess fluid, pulmonary edema may develop. Other factors that increase the outward flux of fluid from the pulmonary vessels include a decrease in the capillary oncotic pressure (hypoproteinemia) and altered microvascular integrity (Enger, 1989). Therefore, an increase in PA wedge pressure is associated with an increase in capillary hydrostatic pressure, which may result in an increase in extravascular lung water.

The PA wedge pressure is clinically used as the indirect method for determination of P_{cap}. The P_{cap} may in fact exceed LA pressure, depending on the PVR (Matthay, Wiedemann, & Matthay, 1985). In the presence of an increased PVR. A high P_{cap} may exist despite a normal PA wedge pressure (Wiedemann, 1987). Research by Cope, Allison, Parmentier, Miller, and Taylor (1986), indicated that measurement of the pulmonary capillary pressure, which can be derived from the PA wedge pressure profile, may be a more accurate reflection of hydrostatic pressure. However, this technique has not been tested in patients suffering an MI; therefore, specific recommendations cannot be made.

Clinical examination of the patient has traditionally been used to assess the onset of ventricular failure and increased extravascular lung water. The principal clinical indicators of cardiac failure include tachycardia, a shifted apical beat, third and fourth heart sounds, valvular incompetence, and signs of pulmonary congestion, e.g., increased jugular venous pressure, basilar crackles, and peripheral edema ("Clinical Signs in Heart Failure", 1989). However, use of these clinical signs has not shown to be particularly accurate in differentiating patients with normal or excess lung water (Luepker, Caralis, Voigt, Burns, Murphy, & Warbasse, 1977), and failed to reflect acute cardiovascular deterioration (Rutherford, McCann, & O'Donovan, 1971; McHugh, Forrester, Adler, Zion & Swan, 1972; Luepker et al., 1977). The chest radiograph was found to be the most accurate noninvasive

indicator of abnormal lung water (Rutherford et al., 1971). However,

as noted, these clinical indicators fail to reflect acute changes.

The PA wedge pressure was found to be a more accurate indicator of

cardiac function. Despite the limitations noted above with regard to

PA wedge pressure's ability to accurately reflect P_{cap}, a PA wedge

pressure of 18 torr was found to separate groups classified

radiographically as absent or minimal LV failure from those with more

severe LV failure (McHugh et al., 1972). In addition, a change in

the PA wedge pressure occurred 1 to 24 hours before changes in the

clinical signs of failure (Rutherford et al., 1971; McHugh et al.,

1972; Luepker et al., 1977). Therefore, in patients with the

potential for deterioration in cardiac status, it was recommended to

use the PA catheter to detect these changes in a more accurate and

timely manner. In addition, assessment of the phase delay in

clinical signs compared with the changes in PA pressure must be

undertaken.

Use of the PA catheter to Guide Therapy in Patients with a Myocardial Infarction

As noted, use of PA pressures as indices of left heart pressure

and volume in the presence of a MI is frought with inaccuracy.

However, a complicated MI is still a primary indication for the

placement of a PA catheter (Shah, 1983; Gore, Goldberg, Spodick,

Alpert, & Dalen, 1987; Spodick, 1989; American College of

Cardiology/American Heart Association [ACC/AHA], 1990; Swan, 1990).

Determination of an optimal range for PA wedge pressure in patients with an MI was undertaken by Crexells, Chatterjee, Forrester, Dikshit, and Swan (1973). The optimal ventricular filling pressure was found to be 15 +/- 2 torr. Crexell and coworkers suggested that in patients with a PA wedge pressure less than 14 torr with a normal heart rate and blood pressure, ventricular function was probably adequate. However, in the presence of hypotension and tachycardia, a PA wedge pressure of less than 14 torr suggested relative hypovolemia; and volume replacement with an increase in the PA wedge pressure would be expected to improve cardiac performance. A filling pressure of greater than 18 torr was not associated with an improvement of cardiac performance. A PA wedge pressure of 18 torr was the level that was associated with the development of radiologic indications of pulmonary congestion (McHugh et al., 1972). Therefore, reduction of filling pressure to less than 18 torr was recommended to relieve pulmonary congestion, and improve oxygen exchange.

A clinical profile that related physiologic and hemodynamic performance was developed to guide clinical therapeutics and determine prognosis in patients with an acute MI (Forrester, Diamond, Chatterjee, & Swan, 1976a, 1976b; Forrester, Diamond & Swan, 1977). Measurement of cardiac index (cardiac output divided by body surface area) and PA wedge pressure were compared with clinical presentation, with the subsequent development of hemodynamic and clinical subsets. The clinical subsets were characterized by the presence or absence of

pulmonary congestion (rales, abnormal chest film) and systemic

hypoperfusion (hypotension, tachycardia, confusion, cyanosis,

oliguria) (Forrester et al., 1977). The hemodynamic subsets were

characterized by the level of the PA wedge pressure and cardiac

index. The relationship between the hemodynamic subsets to the

clinical subsets was then determined (Table 1). Subset I was

characterized by absence of pulmonary congestion and systemic

hypoperfusion, with a PA wedge pressure less than 18 torr, and a

cardiac index greater than 2.2 L/min/m^2. Mortality in this group was

approximately 3%. Subset II was characterized clinically by the

presence of pulmonary congestion, but not systemic hypoperfusion,

Hemodynamically, subset II was characterized by a PA wedge pressure

greater than 18 torr, and a cardiac index greater than 2.2

liters/min/m^2. Mortality for subset II was approximately 10%.

Subset III was characterized clinically by the absence of pulmonary

congestion, but the presence of systemic hypoperfusion. The PA wedge

pressure was less than 18 torr, and cardiac index was less than 2.2

L/min/m^2. Mortality in this subset ranged from 18% to 23%. Subset

IV was characterized by the presence of pulmonary congestion and

systemic hypoperfusion. The PA wedge pressure was greater than 18

torr, and the cardiac index less than 2.2 L/min/m^2. Mortality in

subset IV was greater than 50%.

As illustrated in Figure 6, there is a relationship between PA

wedge pressure and the presence or absence of pulmonary congestion,

and between cardiac index and presence or absence of systemic

Table 1.

<u>Clinical and hemodynamic subset classification of acute myocardial</u>

<u>infarction</u>. From "Medical Therapy of Acute Myocardial Infarction by Application of Hemodynamic Subsets" by J.S. Forrester, G. Diamond, G. Chatterjee, and H.J.C. Swan, 1976, <u>New England Journal of Medicine</u>, <u>295</u>(23), p. 1361. Copyright 1976 by the New England Journal of Medicine. Adapted by permission.

SUBSET	PAWP >18 torr	CI > 2.2 L/min/m^2	PULMONARY CONGESTION	PERIPHERAL HYPOPERFUSION	MORTALITY
I	NO	NO	NO	NO	1-3%
II	YES	NO	YES	NO	9-11%
III	NO	YES	NO	YES	18-23%
IV	YES	YES	YES	YES	51-60%

No pulmonary or peripheral hypoperfusion

Pulmonary congestion without hypoperfusion

TX: Rest Sedation

TX: Diuretics Vasodilator

Peripheral hypoperfusion without pulmonary congestion

Both hypoperfusion Pulmonary congestion

TX: Volume Pacing

TX: Inotropic Vasodilator IABP Surgery

I II III IV

Cardiac Index (CI) L/minute/m²

3.5 3.0 2.5 2.0 1.5 1.0 .5

5 10 15 20 25 30 35

Pulmonary artery wedge pressure (PAWP) mm Hg

Figure 6. Graphic representation of hemodynamic subsets. The heavy lines represent the separation of subsets at pulmonary artery wedge pressure = 18 torr and cardiac index = 2.2 L/min/m², Tx = treatment, IABP = intra-aortic balloon pump. From Cardiac Nursing (p. 566) by S.L. Underhill, S.L. Woods, E.S. Sivarajan Froeliuner, and C.J. Halpenny (Eds.), 1989, Philadelphia: J.B. Lippincott. Copyright 1989 by J.B. Lippincott. Reprinted by permission.

hypoperfusion. In addition to using the relationship between PA wedge pressure and cardiac index and the development of pulmonary congestion or systemic hypoperfusion, respectively, this relationship can also be used to guide clinical therapies through the development of a ventricular function curve (Forrester et al., 1976a).

Figure 7 demonstrates how the optimal LV filling pressure for each patient can be determined by plotting an indicator of LV stretch versus cardiac work (reflective of Starling's law of the heart). Pulmonary artery wedge pressure is often used as the index of LV stretch, while cardiac output or cardiac index is used as the indicator or cardiac work. A more accurate indicator of cardiac work is the stroke volume or stroke volume index. Use of the stroke volume controls for the independent effect of heart rate on cardiac output (Cowan, 1990).

The basic therapeutic goals in the management of an acute MI are to relieve pulmonary congestion by reducing an elevated PA wedge pressure, to relieve systemic hypoperfusion by increasing stroke volume and cardiac index, and to maintain a balance between myocardial oxygen supply and demand (Forrester et al., 1976b). The major determinants of stroke volume, preload, afterload, and contractility, and heart rate can be modified clinically to achieve these goals.

As noted in Figure 7, a family of ventricular function curves can be created to demonstrate any cardiac function. A depressed myocardium (heart failure) has a curve that is shifted down and to
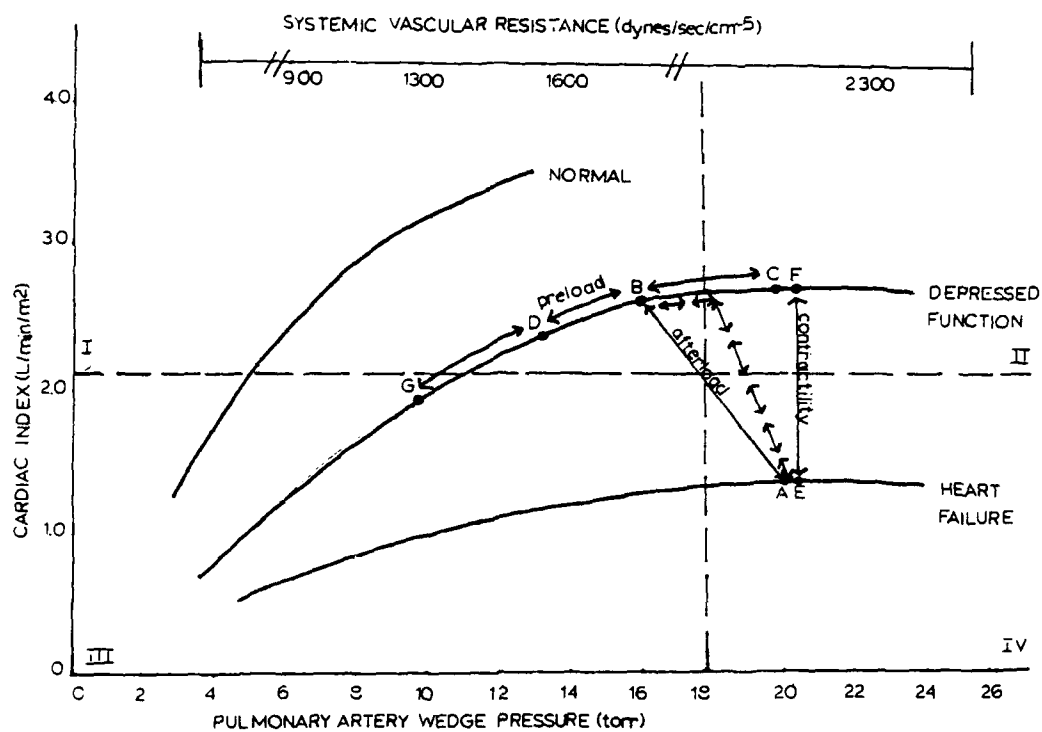
Figure 7. Idealized ventricular function curves represent normal, depressed, and severely depressed function. Point A to Point B reflects a decrease in afterload; Point B to Point A reflects an increase in afterload, Point B to Point C and Point B to to Point D reflect changes in preload; Point E to Point F represents an increase in contractility.

the right, e.g., for any given PA wedge pressure there is a decreased cardiac index. Administration of a positive inotropic agent (dopamine, dobutamine), which increases contractility will shift the curve up (point E to point F), while a negative inotropic agent (propranol, verapamil) will shift the curve down (point F to point E). An alteration in preload (diuresis or volume augmentation) will result in movement up or down a single function curve (point B to point D or point B to point C respectively). An increase in afterload (vasoconstriction) will cause the curve to shift down and to the right (point B to point A), due to an increase in preload and decreased force of contraction. A decrease in afterload will cause the curve to shift up and to the left (point A to point B), due to a decrease in preload and increased force of contraction. The effect of alteration in afterload has minimal effect on preload or cardiac index in the patient with normal contractility; however, in the presence of decreased contractility, i.e., heart failure, changes in afterload may result in clinically important changes in preload and cardiac index. The diagonal shift associated with afterload reduction is related to improvement in contractility, and the subsequent reduction in end-diastolic volume (preload). For example, in a patient with heart failure and hypertension, a reduction in afterload will result in an increase in cardiac index, and a decrease in PA wedge pressure (Figure 7, point A to point B) (Cowan, 1990).

Patients in Subset I do not require specific therapy to improve pump function; however, modification of the determinants of

myocardial oxygen consumption, which include preload, afterload,

contractility and heart rate, may be undertaken to limit the size of

the MI (Braunwald, 1971). For example, reduction of elevated blood

pressure or control of tachycardia may decrease myocardial oxygen

consumption and limit infarct size.

Patients in Subset II are clinically characterized as having

pulmonary congestion without systemic hypoperfusion. The goal of

therapy in this group of patients is reduction of the PA wedge

pressure to a range of 15 to 18 torr (Crexells et al., 1973;

Forrester et al., 1976b). As noted in Figure 7, a reduction in

preload is associated with a decrease in PA wedge pressure, with

minimal change in cardiac index (point C to point B). Diuretic

therapy, which results in a reduction in preload, is generally used

to meet this clinical goal (Swan & Ganz, 1975). If the patient

presents with systemic hypertension, a peripheral vasodilator

(nitroglycerin, nitroprusside) may be effective in reducing PA wedge

pressure without reducing cardiac index, secondary to reduction in

afterload.

Subset III is characterized by systemic hypoperfusion without

pulmonary congestion. The goal of therapy is improvement of the

cardiac index with the resultant relief of the signs and symptoms of

systemic hypoperfusion and without an excessive increase in

myocardial oxygen consumption or the development pulmonary

congestion. Volume infusion is recommended if the filling pressure

(preload) is decreased. The goal of volume augmentation is to raise

ventricular filling pres.ure (PA wedge pressure) to approximately 18

torr (Crexells et al., 1973; Swan & Ganz, 1975). Volume infusion

results in an increase in PA wedge pressure and cardiac index (point

G to point D). The increase in cardiac index is the result of

increased contractile force secondary to increased end-diastolic

fiber length (Starling's law of the heart). If the patient has a

normal stroke volume with a low heart rate, therapy is aimed at

increasing heart rate by use of a vagolytic (atropine), or a positive

chronotropic agent (epinephrine, isoproterenol), or pacemaker

therapy. Caution must be used when increasing heart rate or preload,

because of the effect of heart rate and ventricular chamber size on

myocardial oxygen consumption (supply and demand).

Subset IV is characterized by pulmonary congestion and systemic

hypoperfusion. Patients in this subset are often classified as

manifesting cardiogenic shock (Swan, Forrester, Diamond, Chatterjee,

& Parmley, 1972). The goal for therapy in this group of patients is

to increase cardiac index and decrease PA wedge pressure. Afterload

reduction, by use of a vasodilator (nitroprusside) or a mechanical

assist device (intra-aortic balloon pump), results in an increase in

cardiac index and a decrease in PA wedge pressure (point A to point

B). Use of vasodilator therapy has the potential negative effect of

decreasing coronary perfusion with a resultant worsening of

myocardial ischemia. Because of the potential hypotensive side

effects of vasodilator therapy, the concomitant use of an inotropic

agent, with a resultant upward shift of the ventricular function

curve (point E to point F), and a vasodilator, with a decrease in PA wedge pressure and a minimal change in cardiac index, were recommended to maintain cardiac output and systemic blood pressure. The net result of this concomitant therapy is demonstrated by a shift from point A to point B. Again, caution must be taken when using an inotropic agent in a patient with an imbalance in myocardial supply and demand.

A clinical example of the use of the ventricular function curve is useful to demonstrate its efficacy in guiding clinical therapy. (Figure 8).

Profile: Mr Jones, a 65 year old (75 Kg, BSA = 1.9 m^2), was found collapsed at home. On admission to the Emergency Room, he was confused and restless, skin was cool and clammy. An electrocardiogram demonstrated an acute anterolateral MI. He was tachypneic, with bibasilar crackles. Cardiac auscultation revealed an S3 and S4. Initial vital signs: arterial blood pressure 80/40/53; heart rate 125 beats/minute; respiratory rate 30; and slightly labored. He was admitted to the coronary care unit (CCU) where a PA catheter was placed (see Table 2 for initial hemodynamic profile).

As demonstrated in Figure 8, Mr Jones' initial hemodynamic parameters (point A) are consistent with Forrester's subset IV (cardiogenic shock). Recommended therapy for patients in subset IV includes use of an inotropic agent (point A to point B), and concomitant vasodilator therapy (point B to point C), with a

Table 2.

Case study hemodynamic data.   BP = blood pressure; S = systolic; D =

diastolic; M = mean.

PARAMETERS	INITIAL PROFILE 0800	AFTER THERAPY 1000
BLOOD PRESSURE (S/D/M)	80/40/53	100/60/73
HEART RATE (beats/min)	125	105
RIGHT ATRIAL PRESSURE (mean)	10	7
PULMONARY ARTERY PRESSURE (S/D/M)	38/28/33	32/21/26
PULMONARY ARTERY WEDGE PRESSURE (mean)	22	17
CARDIAC OUTPUT/INDEX (L/min)/(L/min/m^2)	2.8/1.5	42/2.2
SYSTEMIC VASCULAR RESISTANCE(dynes/sec/cm^{-5})	1228	1257
URINE OUTPUT (ml/hr)	0	30
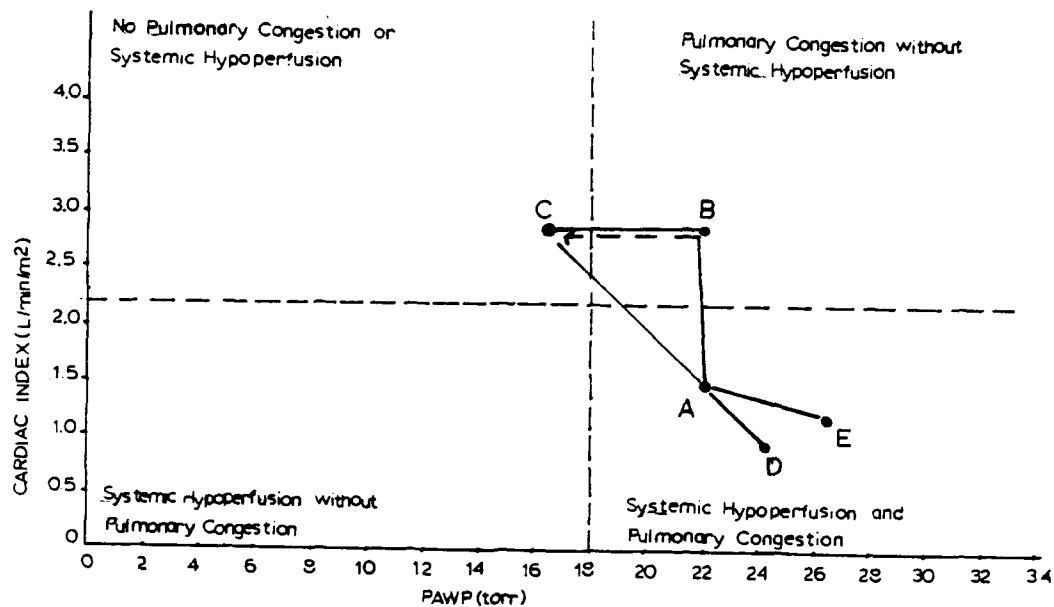
Figure 8. Ventricular function curve reflecting decrease in preload (PA wedge pressure) and increase in cardiac index. Point A to Point B reflects an increase in contractility; Point A to Point C a decrease in afterload; Point A to Point D an increase in afterload; Point A to Point E an increase in preload, and Point B to Point C a reduction in preload.

resultant increase in cardiac index, and a reduction in PA wedge
pressure (point A to point C). Without the use of PA pressure
monitoring, erroneous therapy, such as vasoconstrictor therapy (point
A to point D) or volume loading (point A to Point E) may have been
attempted in an effort to increase blood pressure. In addition to
guiding clinical therapy, PA pressure monitoring and pressure
waveform analysis are useful in determining the exact cause of the
deterioration in cardiovascular function, or the presence of
complications associated with an MI.

The most serious consequence of an MI is the development of
cardiogenic shock. Cardiogenic shock is defined as the "occurrence
of the shock syndrome by reason of primary cardiac dysfunction" (Swan
et al., 1972). This definition excludes hypotension related to pain,
vasovagal response, arrhythmias, drug reactions, or hypovolemia.
Clinically, shock is defined as 1) systolic arterial blood pressure
less than 90 torr. or 30 torr below baseline, and 2) evidence of
reduced peripheral perfusion i.e., urine output less than 20 ml per
hour, impaired mental function, and peripheral vasoconstriction with
cold, clammy skin (Swan et al., 1972). In addition, lactic acidosis
and mixed-venous desaturation will be present (Schreiber, Miller, &
Zola, 1989). Patients with cardiogenic shock are characterized by a
cardiac index less than 2.2 L/min/m^2 and a PA wedge pressure greater
than 18 torr, which is consistent with Forrester's subset IV
(Forrester et al., 1977).

The most common cause of cardiogenic shock in the acute MI patient is LV dysfunction involving more than 40% of the LV wall mass (Lee, 1988; Waller, 1988; Schreiber et al., 1989). Other less frequent causes of cardiogenic shock include extensive RV infarction, and mechanical defects of the right or left heart e.g., rupture of the septum or ventricular free wall or papillary muscle dysfunction or rupture.

Both hemodynamic and clinical presentation are important when differentiating the etiology of cardiogenic shock. Table 3 summarizes the hemodynamic and clinical findings of the various etiologies of cardiogenic shock (Sharkey, 1987; Lee, 1988; Waller; 1988; Daily, 1989; Schriner, 1989). If LV dysunction is the cause of cardiogenic shock, the RA pressure will be normal or elevated and the PA and PA wedge pressures will be elevated.

In a patient with a massive pulmonary embolus, the RA and PA pressures will be elevated, but the PA wedge pressure may be low, normal, or elevated. The elevation in PA wedge pressure may occur secondary to intraventricular septal shift related to RV dilatation (decreased LV compliance). In addition, the pressure difference between the PA diastolic and the PA wedge pressure will be greater than 6 torr, which indicates an elevation in PVR (Bustin, 1986; Darovic, 1987; Sharkey, 1987). There is a high mortality rate associated with a massive pulmonry embolism, and immediate recognition is the key to the prevention of death (Dalen & Alper, 1975).

Table 3.

Clinical and hemodynamic differentiation of etiologies of cardiogenic shock. RA = right atrial,

RAP = right atrial pressure, PA = pulmonary artery, PAD = pulmonary artery diastolic, PAWP = pulmonary

artery wedge pressure, RV = right ventricular, N = no change, ↑ = increased, ↓ = decreased.

PATHOPHYSIOLOGY	RAP	PA	PA WEDGE	PAD-PA WEDGE GRADIENT	CLINICAL FINDINGS
Left ventricular failure	N/↑	↑	↑	N	Pulmonary congestion/edema, S3, S4, increased "a" wave height (due to decreased ventricular compliance). Increased "v" wave due to mitral regurgitation, pulsus alternans
Massive pulmonary embolism	↑	↑	↓/N/↑	↑	Increased RA "v" wave with steep "y" descent due to tricuspid regurgitation, increased alveolar-arterial oxygen gradient, tachypnea, dyspnea, increased pulmonic component of S2, pleuritic chest pain
Right ventricular infarction	↑	↓/↑	↓/↑	N/↑	RAP> PAWP or RAP 1 to 5 torr < PAWP, or RAP > 10 torr, RA tracing with prominent "x" and "y" descent (M configuration), increased jugular venous pressure, systemic venous congestion, right ventricular gallop, split S2, positive hepatojugular reflux, increased RA "a" wave, positive Kussmaul's sign (increased RAP with inspiration) right ventricular S3 or S4
Acute mitral insufficiency	N/↑	↑	↑	Reversed	Acute onset of hypoperfusion and pulmonary edema, dyspnea, tachpnea, increased PAWP "v" wave (early-immediately after T wave), acute increase in PA pressure, split S2, holosystolic murmur at apex, S3 and S4, systolic thrill
Acute ventricular septal defect	↑	↑	Acute↑		Acute hypotension and pulmonary congestion, systolic thrill, holosystolic murmur, acute right heart failure with increased jugular venous pressure, late PAWP "v" wave, oxygen step up of > 10% between right atrium and PA, decreased cardiac output
Acute ventricular free wall rupture	↑	↑	↑		Pulsus paradoxus, negative Kussmaul's sign, RAP tracing with deep "x" descent and attenuated "y" descent, diastolic equalization of all pressures

Right ventricular infarction is most commonly associated with
an inferior wall MI, and results in hemodynamically significant
changes in approximately 5% to 10% of all MIs (Lee, 1988; Waller,
1988,). With a RV infarct the RA pressure is disproportionately
elevated relative to the PA wedge pressure. The presence of a RA
pressure greater than 10 torr and the RA pressure greater than PA
wedge pressure by 1 torr to 5 torr (RA:PA wedge pressure > 0.8), has
a sensitivity of 72% and a specificity of 100% (Lopez-Sendon, Coma-
Canella, & Gamallo, C, 1981; Muirhead, 1989) for the diagnosis of RV
infarction. In addition, the RA tracing demonstrates a prominent "y"
descent, and a equal "x" descent in the presence of mild
noncompliance. In the presence of a severe decrease in RV compliance
the "y" descent is deeper than the "x" descent, resulting in an "M"
or "W" configuration. The PA pressure may be decreased, secondary to
decreased RV stroke volume (preload to the LA), or increased
secondary to RV dysfunction. The PA wedge pressure may be decreased,
due to impaired filling, or increased due to paradoxical septal
displacement, which impairs LV filling (Muirhead, 1989).

Acute mitral insufficiency may occur secondary to papillary
muscle rupture, which is a rare but often fatal complication of an
acute MI, or papillary muscle dysfunction. Papillary muscle
dysfunction is more common with inferior MI, but may occur in an
anterior MI. When the mitral valve fails acutely, a giant "V" wave
may be inscribed on the PA and PA wedge pressure tracings. The "V"
wave is characterized as early as a result of regurgitant blood flow

through the relatively noncompliant left atrium. The PAEDP may be less than the PA wedge pressure due to the presence of the huge "V" wave. However, the presence of a large "V" wave is neither sensitive nor specific to mitral regurgitation, and can also be caused by an acute ventricular septal defect (VSD) (Carley, Wong, Pugh, & Dunn, 1977; Fuchs, Heuser, Yin, & Brinker, 1982; Downes, Hackshaw, Kahl, Santamore, & Little, 1987). The clinical importance of the giant "V" regardless of etiology is the marked increase in pulmonary capillary hydrostatic pressure, with the acute development of pulmonary edema.

Acute VSD occurs in 1% to 3% of acute inferior or anterior MI's, one to seven days after the MI. The rupture of the ventricular septum results in acute volume overload of the right ventricle, due to a left-to-right shunt. There is an acute elevation in RA, PA, and PA wedge pressures. A huge "V" wave may be present on the PA wedge tracing, and is characterized as "late" (well after the QRS complex on the ECG), creating a bifid waveform. The cardiac output is elevated, but only a small portion of the flow is delivered systemically. The key diagnostic criteria for VSD is a step up of greater than 10% in the oxygen saturation between the RA and PA. Ventricular free wall rupture is the second most common hemodynamic cause of death associated with an acute MI. The LV free wall is the most frequent site of rupture, and the rupture usually involves the anterolateral wall. Free wall rupture generally occurs one to three weeks after an acute MI. The result of free wall rupture is the development of a hemopericardium and acute pericardial tamponade.

The hemodynamic characteristics of pericardial tamponade include an increase and equalization of all pressures. Changes in the RA pressure tracing include a deep "x" descent, and a brief "y" descent.

The mean RA pressure decreases during inspiration, which differentiates pericardial tamponade from other conditions that cause equalization of heart pressures (RV infarct and pericardial constriction). As noted in this section, use of the PA catheter in conjunction with clinical assessment aids in the diagnosis of possible etiologies for cardiogenic shock, and serves as a guide for therapeutic decision making.

Technical Factors Affecting PA Pressure Measurement

Before a discussion of the physiologic or pathophysiologic factors that may affect the accuracy of PA pressure measurement, it is important to address the technical factors that affect accuracy of PA pressure measurement. This section discusses use of a standardized reference level, evaluation of the dynamic response of the transducer-catheter system, calibration of the PA catheter system, troubleshooting technical problems, time required for stabilization of PA pressure measurements following position change, and the normal fluctuation in PA pressures.

Reference level.

Correct and consistent referencing of the PA catheter to the left atrium is crucial in ensuring the accuracy of PA pressure measurements. Zeroing the PA catheter system establishes three effects: 1) establishes atmospheric pressure as zero, 2) negates the

weight effect if the catheter tubing system, and 3) ensures that the

pressures that are measured are intracardiac pressures (Gardner,

personal communication, 1991). Winsor and Burch (1945) determined

that the correct reference level (zero point) for the measurement of

venous pressure was the intersection of a frontal plane passing

midway between the posterior surface of the body and the base of the

xiphoid process, and a transverse axis that transected the body at

the junction of the fourth intercostal space and the sternal margin.

The transverse axis was referred to as the phlebostatic axis, and the

horizontal axis as the phlebostatic level. As noted in Figures 9B,

as the patient moves from the flat to the upright position, the

phlebostatic level rotates on the axis and remains horizontal (Winsor

& Burch, 1945; Woods, 1976; Shinn, Woods, & Huseby, 1979).

Frequently, the zero point is referred to as the fourth

intercostal space and the midaxillary line. A study of 81

outpatients was undertaken to determine if the mid-anteroposterior

level (mid-APL) was interchangeable with the midaxillary level (MAL)

(Bartz, Maroun, & Underhill, 1988). The range of differences between

the mid-APL and the MAL was -3.25 cm to +8.25 cm, mean 1.67 cm ($p <$

0.005). The use of the different reference levels would translate

into a pressure difference of up to 6 torr. In a similar study by

Kee, Simonson, Stotts, and Schiller (1987) no statistically

significant difference ($p > 0.05$) was found between the MAL and mid-

APL. However, the latter study did not report the individual

differences between the MAL and mid-APL. In addition, for patients

Fourth
intercostal
space

Lateral margin
of sternum

A

Outermost point
of posterior chest

Outermost point
of sternum

45°

20°

0°

B
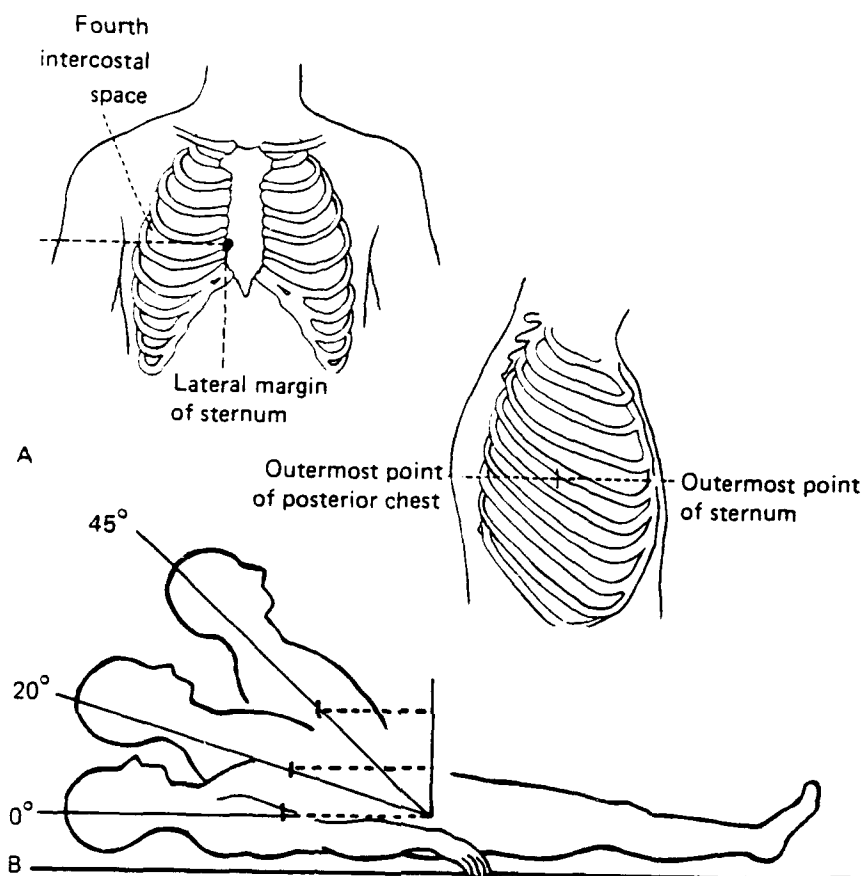
Figure 9A. The phlebostatic axis and phlebostatic level. The
phlebostatic axis is the intersection of the two lines a) a line from
the fourth intercostal space at the point where it joins the sternum,
drawn out to the side of the body beneath the axilla; and B) a line
midpoint between the anterior and posterior surface of the chest.
Figure 9B. The phlebostatic axis is a horizontal line through the
phlebostatic axis. From "Effect of Intermittent Positive Pressure
Ventilation Upon Pulmonary Capillary Wedge Pressure in Acutely Ill
Patients" by J.A. Shinn, S.L. Woods, & J.S. Huseby, 1979, Heart and
Lung, 8(2), p. 324. Copyright 1979 by C.V. Mosby. Reprinted by
permission.

in the supine position, the phlebostatic axis (fourth intercostal
space/mid-APL) has also been confirmed as the level of the mid-left
atrium (Paolella, Dortman, Cronan, & Hasan, 1988). Because the
mid-APL has been validated as the correct venous and mid-left atrial
reference level, use of the MAL should be avoided, to ensure
consistency in PA pressure measurements.

In patients placed in the lateral decubitus position, Paolella
and coworkers (1988) found that in the right lateral decubitus
position the reference level was the fourth intercostal space and the
midsternum, and in the left lateral decubitus position the reference
level was the fourth intercostal space at the left parasternal
border. No specific reference level has been determined for patients
in lateral positions between zero and 90 degrees.

Clinically the zero point is referenced to the air-fluid
interface of the transducer system (Figure 10). If the stopcock is
in-line between the transducer and the catheter hub, the stopcock is
placed at the phlebostatic axis. If the stopcock is on top of the
transducer dome, the stopcock is placed level to the phlebostatic
axis (Quaal, 1984). Placement of the air-fluid interface above the
zero point will result in a measured pressure that is lower than the
actual pressure, conversely, placement of the air-fluid interface
below the zero point results in a measured pressure that is higher
than the actual pressure (an elevation of 1.36 cm is associated with
a pressure decrease of 1 torr; 1.36 cm H_2O = 1 torr). Attention to
exact determination of the zero point, and placement of the air-fluid
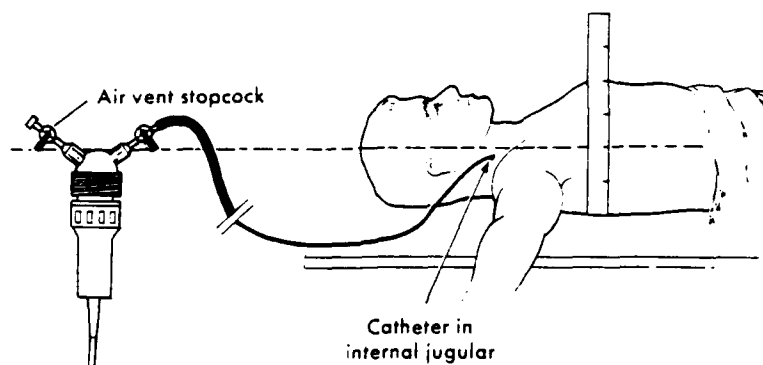
Figure 10. Schematic diagram of correct referencing of the air-fluid interface to the phlebostatic axis From Techniques in Bedside Hemodynamic Monitoring (p. 131) by E. Daily and J.S. Speer, 1989, St. Louis: C.V. Mosby. Copyright 1989 by C.V. Mosby. Reprinted by permission.

interface is crucial in avoiding the introduction of mechanical error into the measurement of PA pressures.

Dynamic response

The catheter-transducer system used for pressure monitoring has been described as an "underdamped, second-order dynamic system" (Gardner, 1981). In such a system two measurements are important with regard to pressure monitoring; natural frequency, and the damping coefficient. The natural frequency refers to how rapidly the system oscillates, and the damping coefficient refers to how quickly the system comes to rest (Gardner, 1981). An optimal dynamic response range is determined by the interaction of the natural frequency and the damping coefficient, and is characterized by a system that is capable of faithfully reproducing all waveforms (Gibbs & Gardner, 1988). The dynamic response characteristics of the catheter-transducer system will be compromised by having a low natural frequency (less than 10 hertz), and a large damping coefficient, which may occur when 1) compliant catheter tubing is used, 2) air bubbles are trapped in the system, 3) a long catheter (PA catheter) or long tubing (greater than 40 inches) are used, or 4) a narrow catheter or narrow tubing are used (Gibbs & Gardner, 1988).

Evaluation of the dynamic response of the catheter-transducer system can be performed by application of a rapid high pressure signal to the transducer dome, i.e., activation of the "fast-flush" system (Marini, 1985). The activation of the "fast-flush" system produces an oscillatory waveform. An optimal dynamic response
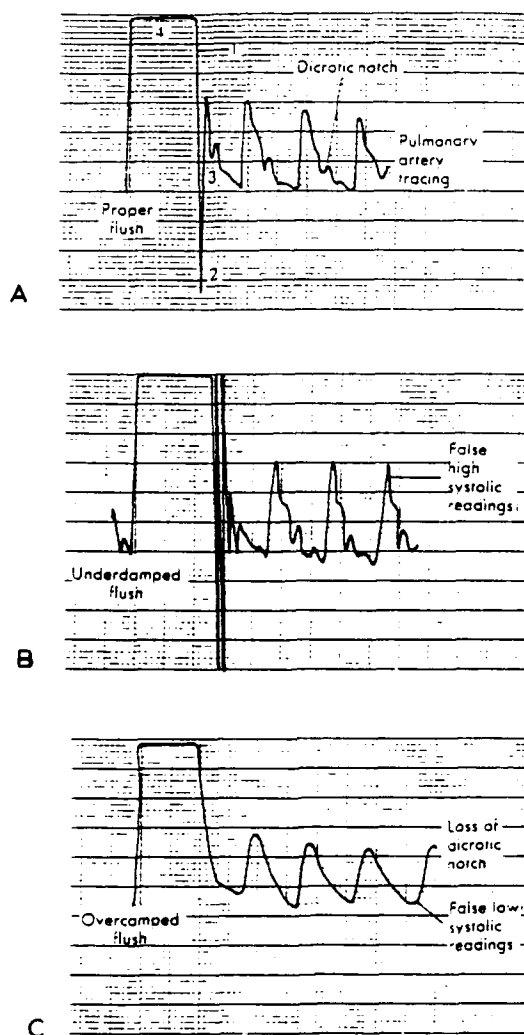
Figure 11A. Proper dynamic response: 1) Two or three rapid oscillations occur from the top to bottom; 2) tracing does not stick on the bottom of graph paper after flush device is released; 3) pattern goes directly back into wave after oscillating, 4) when flush devise is opened, pressure exceeds 200 torr on graph paper (provided the pressure bag is pumped up greater than 200 torr. Figure 11B. Underdamped response. Wide excursions occur above and below the baseline before oscillation comes to rest. Figure 11C. Overdamped flush response. Flush square wave does not fall below baseline once fast flush device is released. From Comprehensive Intra-aortic Balloon Pumping (pp. 225-227) by S.J. Quaal, 1984, St Louis: CV Mosby. Copyright 1984 by C.V. Mosby. Reprinted with permission.

(Figure 11A) is characterized by the presence of two or three rapid oscillations from the top to bottom of the tracing, followed by rapid return to the baseline waveform pattern. In addition, the tracing should not stick on the bottom of the oscilloscope after release of the flush device, and the pressure should exceed 200 torr on the graph paper or oscilloscope (Quaal, 1984).

Gardner (1981) also recommends determination of the natural frequency by measuring the period (distance) of one cycle and applying the following equation:

$$\text{Natural frequency} = \frac{\text{paper speed (mm/sec)}}{\text{one cycle measured in mm}} \text{ Hz}$$

For example in Figure 11A, the paper speed was 25 mm/sec and the distance for one cycle was 1.5 mm; therefore, the natural frequency was approximately 17 hertz. As noted, a system with a natural frequency of less than 10 hertz will not faithfully reproduce the signal. In this case, the natural frequency is adequate for faithful signal reproduction.

An underdamped system transmits all frequency components without a reduction in amplitude (Quaal, 1984). Figure 11B reflects an underdamped system, which demonstates an artificially spiked and exaggerated waveform, with an overestimation of the systolic pressure and an underestimation of the diastolic pressure. The diastolic pressure is less affected than the systolic pressure (Enger, 1989). Possible clinical causes for an underdamped system include respiratory turbulence induced by mechanical ventilation, and

tachycardia. Placement of a mechanical damping device or additional tubing, which increases the damping coefficient, but does not decrease the natural frequency, may be necessary to modify the underdamped waveform (Gardner, 1981; Gardner & Woods, 1989).

Overdamping is the most frequent technical problem associated with PA pressure monitoring. In a study of 282 PA wedge pressure measurements, a 33% rate of technical problems was noted (Morris, Chapman, & Gardner, 1985). Seventy-two percent of the technical problems involved poor dynamic response after a step change or the presence of a damped waveform or both. Overdamping is characterized as a square wave that does not fall below the baseline once the fast-flush device is released (Figure 11C). Overdamping reduces the amplitude of the waveforms, with a diminished dicrotic notch. The pressure measurements reflect a falsely low systolic pressure, and a falsely high diastolic pressure (Quaal, 1984). Causes of overdamping include air bubbles, excessive tubing length (greater than 60 inches), kinks in the tubing or catheter, compliant tubing, clot or fibrin deposition on the catheter tip, greater than three stopcocks, and loose connections (Morris, Chapman, & Gardner, 1985; Gibbs & Gardner, 1988). Careful evaluation and correction of the mechanical factors that cause overdamping will improve the accuracy of PA pressure measurements. Evaluation of dynamic frequency should be completed at least once a shift, following "opening" of the system (zeroing, drawing blood, or changing tubing), and when the pressure

waveform appears damped or distorted (Gardner, 1981; Gibbs & Gardner, 1988).

Calibration.

Calibration involves the application of a known pressure to the transducer system to determine if this pressure is accurately displayed (Enger, 1989). For example, application of 0, 50, or 100 torr of pressure from a mercury manometer to the system should result in a corresponding display on the digital readout of the monitor. In addition, an open-ended water-filled tubing connected to the transducer dome can be use to calibrate the system. The pressure tubing is turned "off" to the patient and "open" to air, and the air-fluid interface is vertically raised a given distance above the transducer (an elevation of 13.6 cm should be reflected on the display as -10 torr). Newer technology pulls on the transducer wires, rather than pushing on them (water or mercury). If the pressure display varies markedly from the expected value, either the gain, scale, or position adjustments of the recording device are incorrectly set, or the transducer/amplifier system is malfunctioning. The latter problem can be evaluated by use of the "self-test" system on the amplifier/display unit (Sears & Heise, 1980; O'Quin & Marini, 1983; Enger, 1989).

Troubleshooting the PA catheter.

As noted the most frequent technical problem associated with PA pressure monitoring is the presence of an overdamped tracing. Other problems that require investigation and resolution include

overwedging or spontaneous wedging of the PA catheter, absence of, or
inability to attain a PA wedge pressure tracing, catheter whip,
inappropriate pressure readings with a proper waveform, and migration
of the catheter into the right ventricle (Runkel & Burke, 1983;
Noone, 1988; Enger, 1989; Gardner & Woods, 1989). Spontaneous
wedging of the catheter in the PA and migration of the catheter into
the right ventricle have potentially serious consequences if not
resolved immediately. The other problems will result in inaccurate
pressure monitoring, and may result in errant therapeutic decisions
and treatment. Table 4 summarizes possible etiologies, indicators,
and recommendations for management of each of these problems.

Pulmonary artery pressure stabilization.

Leppanen (1979) studied the time required for stabilization of
PA pressure measurements following a change in backrest position of
acutely ill patients. The study found that all PA pressure
measurements could be obtained immediately following a position
change of 0 to 20 degrees, without statistically ($p > 0.05$) or
clinically significant differences in pressures. This study was
limited by the small sample size, measurement of pressures over an
entire respiratory cycle, and limited position change. In addition,
while mean changes did not reflect clinically significant changes,
large individual variations did exist. Further research with regard
to stabilization needs to completed in relation to other
interventions including larger position change, and lateral position
change.

Table 4.

Troubleshooting the pulmonary artery catheter

Problem	Etiology	Indicators	Management
Absence of PAWP tracing	Balloon rupture, over or underinflation, improper positioning of PA catheter	Lack of resistance to air injection into air port, blood leaking from balloon port.	1) Allow passive deflation, 2) confirm catheter position with chest radiograph film, 3) have patient cough, 4) label balloon port "Do not wedge" if balloon ruptured
Catheter whip "fling":	Movement of catheter in right ventricle, which accelerates the fluid in the catheter system.	Jagged appearance on pressure tracing	1) Assess dynamic response of catheter-transducer system, 2) add pressure tubing or mechanical overshoot eliminator, 3) record pressures using "maen" if fling fails to resolve
Migration of catheter into right ventricle	Accidental or spontaneous withdrawal of catheter into right ventricle	Decreased diastolic pressure, presence of right ventricular ectopy, loss of dicrotic notch on PA waveform	1) Inflate the balloon, 2) reposition catheter into PA 3) withdraw catheter into RA
Inappropriate pressure with proper waveform	Improper air-fluid interface, inaccurate calibration		1) Check transducer level, 2) recalibrate PA catheter system, 3) rezero system to phlebostatic axis, 4) change scale for readings
Damped curve	Clots in the system, blood in the transducer, air in system, occlusion of catheter tip by balloon or wall of PA, kink in catheter, use of compliant tubing	Decreased amplitude of waveform, decreased systolic pressure, increased diastolic pressure, diminished dicrotic notch	1) Check for intact system, 2) check for air bubbles, 3) check for adequate pressure (300 torr) and volume in the pressure system, 4) locate kinks in the system, 5) aspirate pressure system, 6) flush transducer head, 7) have patient cough, extend arm, or turn onto side, 8) withdraw catheter (verify with physician), 9) use high pressure tubing that is less than four feet in length
Overwedging	Catheter migration	Catheter wedges with less than balloon capacity, elevation of PA wedge pressure waveform that drift- the screen	1) Inflate balloon only enough to obtain a waveform, 2) document volume of air required during initial inflation, 3) catheter may need to be repositioned
Spontaneous wedge (persistent PA wedge)	Catheter migration	acing present ation of balloon	1) Have patient cough, turn onto side, or straighten arm to dislodge catheter, 2) notify physician - anticipate repositioning of PA catheter, 3) maintain continuous PA pressure monitoring

Normal PA pressure fluctuation

Nemens and Woods (1982) studied normal fluctuation in PA

pressures in 26 acutely ill patients. Over a 30-minute period, PA

systolic pressure fluctuated 2 to 7 torr, mean 3.9 torr; PA diastolic

pressure fluctuated 1 to 6 torr, mean 3.4 torr; PA mean pressure

fluctuated 1 to 5 torr, mean 2.8 torr; and PA wedge pressure

fluctuated 0 to 7 torr, mean 3.4 torr. The fluctuations in pressures

were independent ($p > 0.05$) of cardiac output, backrest elevation,

mean PA pressure, or ventilatory support. Therefore, clinically

significant changes in PA pressures were described as 4 torr for PA

diastolic and PA wedge pressures, and 5 torr for PA systolic and PA

mean pressures. Limitations of this study included the small sample

size, measurement of pressures over an entire respiratory cycle, and

limited time period. In addition, the patients in this study, all

had PA wedge pressures between 5 and 18 torr, and therefore, did not

reflect extremes. This study was important because it noted that

pressure changes of 4 torr or less may simply be a reflection of

normal fluctuations in PA and PA wedge pressures. However,

assessment of each individual was still necessary.

Attention to the technical factors of reference level, dynamic

response, calibration, and troubleshooting the PA catheter system, as

well as the physiologic variables, stabilization period and normal

fluctuation will increase the accuracy of PA pressure measurements.

As will be discussed in the sections regarding the effects of

positioning and pulmonary factors on PA pressure measurements, many

studies do not address these factors; therefore, clinical

implications from the study findings are limited.

Pulmonary Effects on PA Pressure Monitoring

Numerous pulmonary variables can affect the accuracy of PA

pressure measurement. This section addresses the following variables

that affect PA pressure measurements: 1) the interaction of pulmonary

arterial (P_a), pulmonary venous (P_v), and alveolar (P_A) pressures on

blood flow in the lungs, 2) respiratory variations during spontaneous

and mechanical ventilation, 3) the effects of mechanical ventilation

and positive end-expiratory pressure (PEEP), and 4) the effect of PA

catheter position relative to the left atrium. In addition,

technical factors including 1) measurement on versus off the

ventilator, 2) measurement at end-expiration, and 3) methods to

adjust PA pressure readings for the effects of PEEP are be discussed.

Pulmonary blood flow.

West, Dollery and Naimark (1964) determined that the lungs have

three physiologic zones of blood flow, depending on the interaction

of P_a, P_v, and P_A pressures as demonstrated in Figure 12. Zone 1 is

characterized by an absence of blood flow. Alveolar pressure exceeds

both arterial and venous pressures ($P_A > P_a > P_v$), resulting in the

collapse of the pulmonary vasculature. Zone 2 is characterized by

intermittent blood flow. Arterial pressure exceeds both alveolar and

venous pressures ($P_a > P_A > P_v$). The relationship between the venous

and alveolar pressure is variable. Because the pulmonary vasculature

is collapsible, when alveolar pressure exceeds venous pressure the
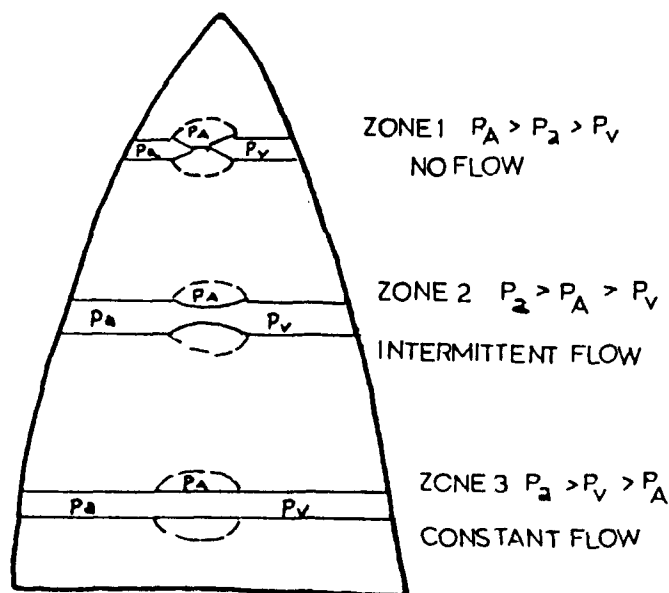
Figure 12. Physiologic zones of the lung. P_a = pressure arterial; P_v = pressure venous; P_A = pressure alveolar. From "Distribution of Blood Flow in Isolated Lung, Relation to Vascular and Alveolar Pressures" by J.B. West, C.T. Dollery, & A. Naimark, 1964, J Appl Physiol, 19(4), p 713. Copyright Journal of Applied Physiology, 1964. Adapted with permission.

vessel collapses. In this case, the driving force for flow is the difference between the perfusing pressure (P_a) and the alveolar pressure (P_A). This area of the lung is reflective of a Starling resistor. Flow in Zone 2 can be altered either by an increase in alveolar pressure or a decrease in venous pressure. Zone 3 is characterized by arterial and venous pressures that both exceed alveolar pressure ($P_a > P_v > P_A$). Blood flow in this zone is constant, and is determined by the difference between arterial inflow (P_a) and venous outflow (P_v). Flow through the upright human lung increases in a linear fashion from the apex to the base, due to the hydrostatic pressure gradient, with flow greatest at the base.

The variable flow in the lungs has an impact on the correct functioning of the PA catheter, which requires an open blood-filled column between the wedged PA catheter and the left atrium. If the PA catheter is placed into either Zone 1 or 2 where flow is absent or intermittent, this requirement will not be met. A PA catheter in Zone 1 will reflect alveolar pressure, but will not reflect vascular pressure. A PA catheter placed in Zone 2 will reflect PA pressure when the balloon is deflated, but will reflect alveolar pressure when wedged, causing an artificially high PA wedge pressure. Optimal placement of the PA catheter is in Zone 3 where flow is continuous. However, Zone 3 can be converted to Zone 2 or 1 by elevation of alveolar pressure (PEEP therapy), or with a decrease in venous pressure (extracellular fluid deficit) (West et al., 1964; O'Quin & Marini, 1983). The importance of the placement of the catheter into

Zone 3 will become more apparent during the discussion of the affect

of PEEP on PA pressure measurement accuracy.

On versus off mechanical ventilation.

Six studies (Appendix B) could be found that determined the

effect of positive-pressure ventilation with or without PEEP on the

accuracy of PA pressure measurement. Davison, Parker, and Harrison

(1978) found no significant difference in mean PA wedge pressure in

29 ICU patients, on mechanical ventilation without PEEP ($p > 0.5$),

compared with off ventilator pressures. While Shinn, Woods, and

Huseby (1979), and Grose and Woods (1981) found statistically but not

clinically significant changes in mean PA pressures on and off

mechanical ventilation ($p < 0.001$ and $p < 0.01$ respectively). In the

latter two studies, a small number of patients demonstrated

individual variations that were greater than the expected

fluctuation. A limitation of the study was that pressures were not

measured at end-expiration, but were averaged throughout the

respiratory cycle. Therefore, in patients receiving mechanical

ventilation without PEEP, removal from the ventilator for PA pressure

measurements was generally not warranted, however, individual

variations need to be assessed.

The study by Shinn and coworkers (1979) also found a large mean

difference between PA diastolic and PA wedge pressures on [-5.2,

($p < 0.01$)] and off [-3.9, ($p < 0.05$)] mechanical ventilation. Based

on these results the author's recommended that PA diastolic pressure

should not be used in place of PA wedge pressure as an index of LVEDP.

Four studies have evaluated the effect of removal from mechanical ventilation when PEEP was used. Davison and colleagues (1978) found no significant difference in mean PA wedge pressure ($p > 0.05$) on and off mechanical ventilation. Van Sciver studied 14 patients (12 with LV dysfunction) on and off the ventilator. In the 12 patients with LV dysfunction there was a significant increase in PA systolic and diastolic ($p < 0.001$), and PA wedge pressure ($p < 0.05$) off the ventilator. These changes did not occur in the patients with normal LV function. Gershan (1983) found a significant difference ($p < 0.01$) in mean PA wedge pressure off mechanical ventilation with PEEP greater than 10 cm H_2O. In addition, mean PA systolic and PA diastolic pressures increased off mechanical ventilation when PEEP was 16 to 20 cm H_2O, but decreased when PEEP was less than 15 cm H_2O. Findings from this study are limited due to the use of group data. In patients with adequate blood volume (less than 1000 ml deficit) PA pressures on and off mechanical ventilation with PEEP less than 10 cm H_2O were not different (Lookinland, 1989a, 1989b). However, in patients with a blood volume deficit greater than 1000 ml, PA pressures were lower off mechanical ventilation. The studies by Lookinland are limited by the lack of statistical analysis of the data presented. The four studies, related to the effect of mechanical ventilation, evaluated four different patient populations, and found that the level of PEEP (greater than 10 cm H_2O), LV

dysfunction, and blood volume all affect a patient's response to removal from mechanical ventilation. The presence of these variables needs to be addressed when evaluating the accuracy of PA pressures on and off mechanical ventilation.

Riedinger, Shellock, and Swan (1981) suggested that removal of the patient from mechanical ventilation does not provide an accurate description of the true cardiopulmonary pressures that the patient is subjected to as a result of positive pressure ventilation. Mechanical ventilation and PEEP may compromise the cardiovascular system, but these changes may not be apparent if the patient is removed from the ventilator for PA pressure measurements. As noted by Van Sciver (1982), removal may cause further dysfunction in a patient with LV failure.

In addition to the effect on the heart, Lookinland (1989a, 1989b) addressed the effect of removal from mechanical ventilation on oxygen delivery. Arterial oxygen tension in 30 cardiac surgery patients was significantly lower ($p < 0.05$) than baseline throughout removal from mechanical ventilation, and for 30 minutes after return to the ventilator. Therefore, removal of the patient from the ventilator in an attempt to increase PA pressure measurement accuracy may further compromise the patient's dysfunctional status.

Effect of the ventilatory cycle: spontaneous versus mechanical ventilation.

Airflow into the lung during spontaneous inspiration is due to a decrease in pleural pressure relative to atmospheric pressure

(Figure 13). During quiet inspiration alveolar pressure is approximately -1 cm H_2O relative to atmospheric, resulting in a pressure gradient for airflow (Campbell & Greenburg, 1986). During passive exhalation alveolar pressure rises to approximately +1 cm H_2O relative to the atmosphere. These changes in pleural pressure are transmitted to cardiovascular structures in the thorax, and are reflected by changes in PA pressure measurements during inspiration and expiration (Figure 14).

During spontaneous ventilation PA pressures decrease during inspiration, and rise during exhalation (Figure 14). During mechanical ventilation the pressure gradient for airflow is reversed. Positive pressure occurs during inspiration with a resultant increase in pleural pressure, which is transmitted to the cardiac structures. Pulmonary artery pressures in the mechanically ventilated patient rise during inspiration (the opposite of spontaneous ventilation), and decrease during exhalation (Figure 15).

Several strategies have been suggested to correct this respiratory variation (Riedinger, Shellock, & Swan, 1981; O'Quin & Marini, 1983; Wild & Woods, 1985) including breath holding, removal from the ventilator, reading mean pressures, pressure averaging, and reading the pressure at end-expiration. A limitation of breath holding is the inadvertent performance of a Valsalva's maneuver with transmission of pressure to the chest. As discussed, the removal of the patient from the ventilator may cause cardiopulmonary embarrassment, and the pressures will fail to reflect the patient's
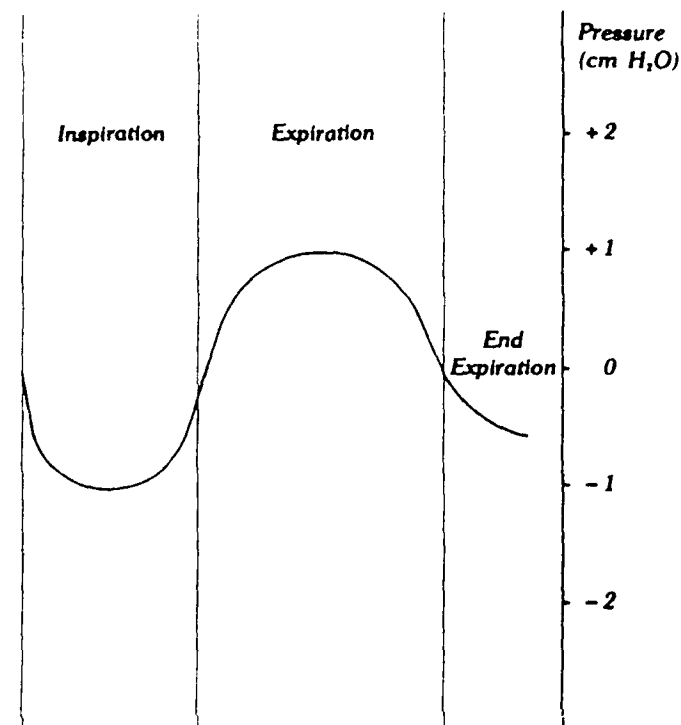
Figure 13.   Intrapulmonary pressure changes relative to atmospheric pressure during spontaneous inspiration and expiration.   From "Reading Pulmonary Artery Wedge Pressure at End-Expiration" by M.L. Campbell and C.A. Greenburg, 1988, Focus on Critical Care, 15(2), (p. 61).   Copyright 1988 by C.V Mosby Company.   Reprinted by permission.

ECG

PAWEDGE PRESSURE

a v

expiration

RESPIRATORY TRACE

inspiration   expiration
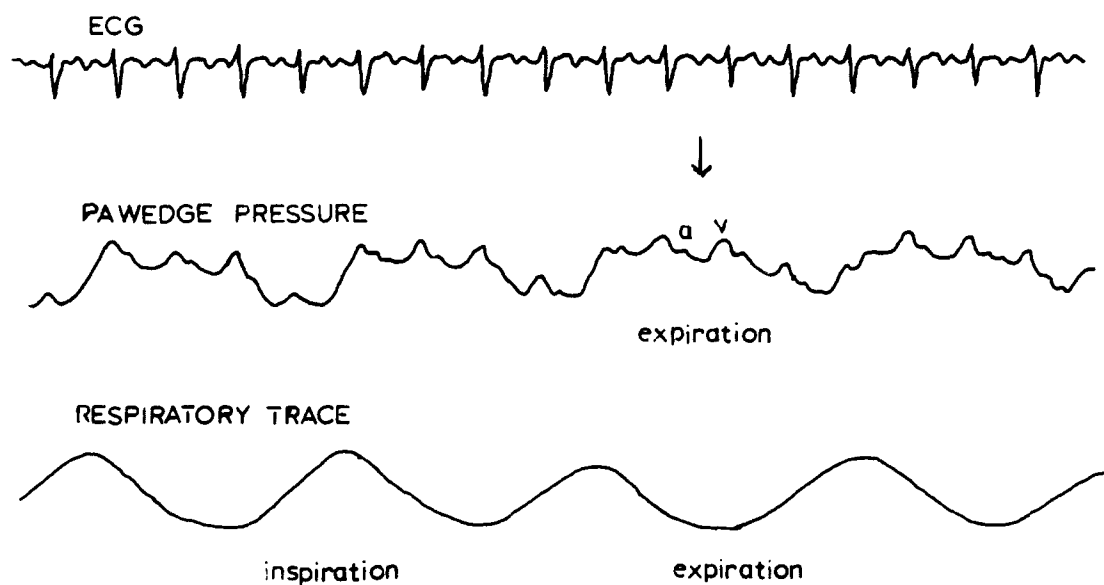
Figure 14. Variation in pressure waveform due to spontaneous inspiration and expiration. Pressure is read at the end of expiration (arrow).
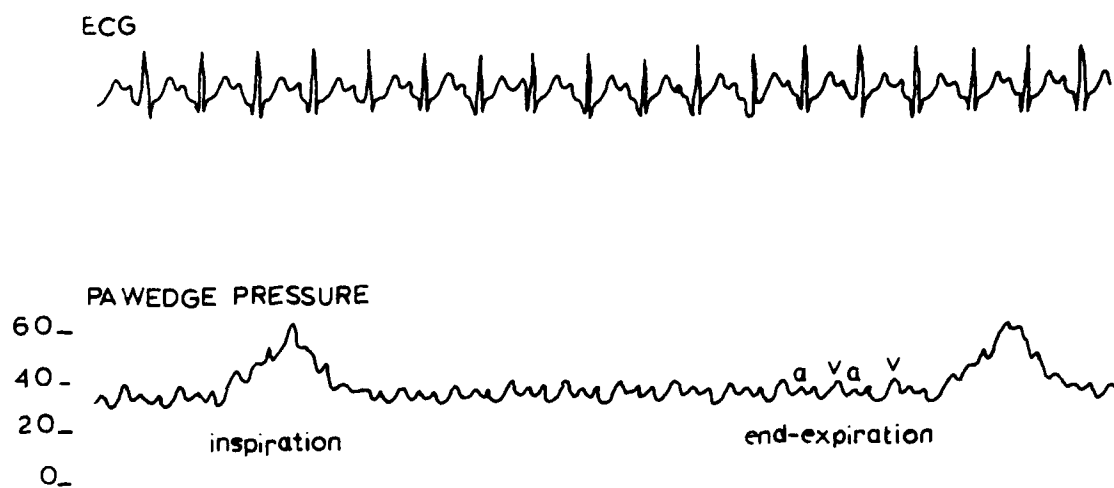
Figure 15.   Variation in PA wedge pressure waveform due to mechanical ventilation.   Pressures are read at end-expiration.

status while on the ventilator.

As noted above, changes in PA pressure occur as a result of changes in pleural pressure during ventilation. At end-expiration, pleural pressure equals atmospheric pressure, and no air flow occurs. Therefore, pleural pressure at end-expiration is constant and does not affect intracardiac pressures. This finding was confirmed in a study by Berryhill, Benumof, and Rauscher (1978) that simultaneously recorded PA and airway pressures in ten mechanically-ventilated patients. Using this method, pulmonary vascular pressures were consistently within a 2 torr range. Wild and Woods (1985) evaluated three methods for reading PA wedge pressure waveforms with respiration. Method 1 calculated the area under the pressure curve, method 2 calculated an arithmetic mean, and method 3 measured trough diastole, representing end-expiration (Figure 16). Clinically and statistically significant differences between PA wedge pressure read at end-expiration (method 3) and method 2 and 1 ($p < 0.01$) occurred, while no signficant difference existed between methods 1 and 2 ($p > 0.01$). While end-expiration (method 3) is considered to be the most accurate, this study highlighted the need to be consistent in the method used to read PA wedge pressure.

Another factor that may alter the accuracy of PA pressure readings with respiratory variation is the use of digital versus analog (graphic) recordings. Maran (1980), Cengiz, Crapo, and Gardner (1983), and Levine (1985), all demonstrated that digital readings were unreliable because of the unselective nature of

electrical averaging. In spontaneously breathing patients Cengiz and coworkers (1980) found that mean PA pressure ($p < 0.01$), and PA wedge and PA diastolic pressures ($p < 0.001$) were significantly lower than analog data, with a mean difference of 10 torr for all values. In patients receiving intermittent mechanical ventilation, the digital PA systolic pressure was significantly higher ($p < 0.05$) than analog data (mean difference 4.8 torr), and digital PA diastolic pressure was significantly lower ($p < 0.001$), with a mean difference of 12 torr. In addition digital mean PA pressure ($p < 0.05$) and PA wedge pressure ($p < 0.001$) were statistically, but not clinically lower than analog data. In the assist/control mode digital PA systolic ($p < 0.005$) and PA wedge ($p < 0.001$) pressure were statistically higher, and PAD was clinically and statistically ($p < 0.001$) lower than manually measured data. The PA mean pressure was not affected. These findings were confirmed in three cases studied by Maran (1980). It was recommended not to use digital data unless it was compared with analog data and found to be similar (Gershan, 1983). If digital data are the only available method for measuring PA pressures, during controlled mechanical ventilation PA wedge pressure should be measured with the diastolic pressure mode (lowest pressure), during assisted ventilation PA wedge pressure should be read using the digital mean, and during spontaneous ventilation the systolic pressure mode (highest pressure) mode should be used (Silverman, Eppler, Pitman, & Patz, 1984; Marini 1985). Newer monitoring equipment allows for the selection of specific waveforms which should

eliminate this discrepancy; however, this has not been verified
through systematic evaluation.

Mechanical ventilation with PEEP.

As noted in the discussion of the physiologic zones of the
lung, a change in alveolar pressure may alter the relationship
between PA wedge and LA pressure, due to compression of the pulmonary
vasculature. This section discusses the possible causes of a PEEP-
induced decline in cardiac function, the effect of PEEP on the
accuracy of PA wedge pressure readings, the effect of catheter
position on the accuracy of PA wedge pressure measurements in the
patient receiving PEEP (Appendix C), and suggested methods for
compensating for the PEEP effect.

The administration of PEEP has been found to be effective in
decreasing hypoxemia in patients with adult respiratory distress
syndrome (ARDS) (Weisman, Rinaldo, & Rogers, 1982). However, an
unwanted side effect of PEEP therapy is a decrease in cardiac output.
Jardin and coworkers (1981) studied ten patients with ARDS who
received PEEP in 5 cm increments from 0 to 30 cm H_2O. At levels of
PEEP greater than 10 cm H_2O PA wedge pressure was consistently
greater than LVEDP, and LV dimensions decreased from baseline. At
greater than 15 cm H_2O of PEEP, the radius of septal curvature
decreased at both end-diastole and end-systole indicating a leftward
shift of the interventricular septum. The authors hypothesized that
the decrease in cardiac output was mediated by acute RV loading with
septal flattening and bulging into the left ventricle, with

subsequent impairment of LV function. This study was limited by the small sample size, which only included patients with acute ARDS, but no previous cardiac or pulmonary dysfunction. In addition, the changes mediated by the interventricular interdependence only occurred at higher levels of PEEP.

Qvist, Pontoppidan, Wilson, Lowenstein, and Laver (1975) studied 18 dogs to determine the effect of volume loading on hemodynamic parameters following the administration of PEEP. In contrast to the study by Jardin and coworkers (1985), these researchers found that the decrease in cardiac output was not due to impaired ventricular function (as was the case with septal displacement), but was associated with a decrease in ventricular filling pressure, which could be offset by volume expansion. Of particular interest was that the acute removal of PEEP following volume expansion resulted in rebound hypervolemia (autotransfusion effect), which could further compromise the patient. The effect of ventricular compromise due to rebound hypervolemia was confirmed clinically in humans by Van Sciver (1982). The study by Qvist and coworkers (1975), only evaluated the effect of 12 cm H_2O PEEP, while the Jardin study looked at PEEP across a wide range. In addition, prior to initiation of the study, the dog models all had normal lungs and heart.

Similar results were found in a study of six healthy baboons (Zarins, Virgilio, Smith, & Peters, 1977). Positive end-expiratory pressures was applied (0-20-0), in 5 cm increments while

intravascular volume was altered. The subset of hypovolemic animals
(n = 5) all demonstrated a decrease in cardiac output with the
application of as little as 5 cm H_2O of PEEP; therefore, further
evaluation of this group was not completed. In the normovolemic
animals cardiac output fell with increased PEEP and was significantly
decreased at 10 cm H_2O (p < 0.01), 15 cm H_2O, and 20 cm H_2O PEEP
(p < 0.001). At greater than 15 cm H_2O of PEEP PA wedge pressure no
longer reflected LA pressure. Volume expansion resolved the PA
wedge-LA pressure discrepancy at all levels of PEEP. These findings
tend to support decreased intravascular pressure as a cause of
depressed cardiac function.

These studies are of interest because they indicated that two
mechanisms altered cardiac function depending on the level of PEEP
applied. Clinically, volume expansion may be the method of choice
for treating a decreased cardiac output when low levels (less than
10 cm H_2O) of PEEP are applied, while at higher levels of PEEP this
would exacerbate the patient's condition due to the interdependence
of the right and left ventricle. In addition, none of the studies
found altered contractility as a cause of PEEP induced cardiac
depression.

Lozman (1974) studied five cardiac surgery patients and found
in 9 of 32 data sets (all nine at PEEP greater than 10 cm H_2O) that
PA wedge pressure changes did not reflect changes in LA pressure, but
below 10 cm H_2O PA wedge pressure was a good indicator or LA
pressure. Similar findings occurred in a study of the effect of 5 cm

H_2O increase in PEEP (0-20 cm H_2O) in six healthy baboons (Hobelmann, Smith, Virgilio, Shapiro, & Peters, 1974). Clinically significant differences were noted between PA wedge pressure and LA pressure for all animals at 15 cm H_2O of PEEP (mean difference = 5 torr), and 20 cm H_2O of PEEP (mean difference = 10 torr). Several possibilities for this discrepancy were presented: 1) Starling-resistor phenomenon caused by a PEEP induced increase in alveolar pressure, 2) increased PVR, and 3) unequal balloon inflation. The latter factor was confirmed in *in vitro* and *in vivo* dog studies. All the patients in this study (*N* = 5) developed LV failure when receiving greater than 10 to 15 cm H_2O of PEEP.

To function accurately, the PA catheter must have a continuous fluid channel between the catheter tip and the left atrium. The use of PEEP may compress the pulmonary vessels, and alter the accuracy of the pressure readings. Six studies have evaluated the effect of PA catheter placement in various physiologic zones on the accuracy of PA pressure measurements (Appendix C). Two dog studies, Roy, Powers, Feustel, and Dutton (1977), and Kane, Askanzi, Neville, Mon, Hanson, and Webb (1978) studied the effect of incrementally increased PEEP on PA wedge and LA pressure difference when the PA catheter was placed above or below the left atrium. In the study by Roy and coworkers, significant (*p* < 0.01) differences between PA wedge and LA pressures occurred at 10 cm H_2O PEEP in the catheter placed above the left atrium, while a statistically (*p* < 0.01) but not clinically significant difference between PA wedge pressure and LA pressure

occurred at PEEP greater than 15 cm H_2O in the catheter placed below the left atrium. The findings with regard to the placement of the catheter above the left atrium were confirmed by Kane and coworkers. However, Kane and coworkers did not find a significant difference between PA wedge pressure and LA pressure at any level of PEEP ($p > 0.05$).

Two studies using cardiac surgery patients have also evaluated the effect of catheter position on PA pressure measurement accuracy (Shasby et al., 1981; Rajacich, Burchard, Hasan, & Singh, 1989). In the study by Shasby and coworkers (1981), in the catheter placed below the left atrium ($n = 8$), no significant difference ($p > 0.20$) between PA wedge-LA pressure occurred until PEEP reached 11 torr (15 cm H_2O), and in the study by Rajacich and coworkers no significant difference ($p > 0.01$) occurred over a range of 0 to 20 cm H_2O in the catheter below the LA ($n = 12$). In the catheter placed above the left atrium ($n = 5$), a significant PA wedge-LA pressure difference ($p < 0.05$) occurred above 15 cm H_2O of PEEP in the study by Rajacich and coworkers (1989), while Shasby and coworkers found that the PA wedge-LA pressure difference was dependent on the absolute LA pressure, a factor not addressed by Rajacich.

Hasan, Malanga, Braman, Carrao, and Most (1984) simultaneously measured PA wedge pressure and LA pressure using PA catheters placed in the right and left lungs of 10 pigs. The pigs were positioned into the right or left lateral position. Pulmonary artery wedge pressure that was measured in the lateral position, with the catheter

tip positioned vertically below the left atrium more accurately ($p < 0.05$) reflected LA pressure over 0 to 20 cm H_2O PEEP, than did the PA wedge pressure measured in the catheter positioned at or above the left atrium.

A second report using the same pig population (Malanga, Hasan, Braman, Carrao, & Most, 1984) again found no difference between PA wedge pressure and LA pressure in the catheter placed below the left atrium up to 20 cm H_2O of PEEP, while PA wedge pressure was significantly ($p < 0.001$) greater than LA pressure in the catheter placed above the left atrium at greater than 10 cm H_2O of PEEP. These two reports demonstrate the importance of placement of the catheter into zone 3 to ensure the increase the accuracy of pressure measurement.

Another factor that was found to affect PA pressure measurement accuracy was the presence or absence of lung disease. Tooker, Huseby and Butler (1978), Berryhill and Benumof (1979), and Hasan, Weiss, Braman, and Hoppin (1985) evaluated the effect of lung injury and PA catheter placement on PA pressure measurement accuracy. All three studies found that lung injury increased PA wedge and LA pressure correlation across all levels of PEEP, regardless of catheter position, but placement of the catheter in the dependent position further increased the correlation and pressure measurement accuracy.

Vascular volume was also been found to affect pressure measurement accuracy when PEEP was applied (Kane et al., 1978; Shasby et al., 1981). Hypovolemia induced by bleeding, with the catheter

above the left atrium (n = 5) was associated with a significant

difference (p < 0.05) between PA wedge and LA pressures at 10 cm H_2O

of PEEP (Kane, 1978). The effect of hypovolemia was confirmed by

Shasby and coworkers (1981), who found a significant difference

(p < 0.05) in PA wedge pressure and LA pressure occurred in the

catheter above the LA (n = 6), when LA pressure was less than 5 torr,

compared with catheters placed above the LA, when LA pressure was

greater than 5 torr. These findings were consistent with conversion

of Zone 3 to Zone 2 or 1 by lowering the venous pressure or raising

the alveolar pressure, thus compressing the vascular bed. Again,

positioning the catheter in a dependent position increased the

reliability of PA wedge pressure as an indicator of LA pressure,

regardless of vascular volume.

Finally, controlled versus spontaneous ventilation was found to

affect the accuracy of PA pressure measurements when PEEP was applied

(Berryhill & Benumof, 1979). Spontaneous ventilation in six dogs was

found to provide protection over a wide range of PEEP levels,

regardless of the presence or absence of lung injury. However,

during continuous positive-pressure ventilation with PEEP, both

catheter position and the degree of lung injury significantly

affected the accuracy of PA pressure measurements (p < 0.01), in a

manner consistent with the studies already reviewed.

The studies of the effect of PEEP and catheter position on the

accuracy of PA wedge pressure as an indicator of LA pressure have

resulted in similar findings: 1) at PEEP less than 10 cm H_2O, PA

wedge pressure is a good indicator of LA pressure, 2) decreased compliance minimizes the effect of increasing PEEP on PA wedge pressure, 3) correct positioning of the PA catheter below the left atrium (in both supine and lateral position) increases the reliability of PA wedge pressure, 4) vascular volume affects the accuracy of PA pressure measurements during PEEP therapy, and 5) spontaneous breathing during PEEP therapy does not affect PA pressure measurement accuracy, but continuous positive-pressure ventilation does.

Further research is needed in the following areas 1) use of a more heterogenous human population, including patients with impaired cardiac function, 2) assessment of varying degrees of lateral position changes on the the accuracy of the PA wedge pressure with the catheter above and below the LA, and 3) the effect of intermittent mechanical ventilation with PEEP on PA pressure measurement accuracy.

As noted, positioning the catheter in the dependent position increases the accuracy of PA wedge pressure as an indicator of LA pressure. Several other methods have been suggested to correct for the effects of PEEP on PA pressures including estimation of extramural pressure, and use of an esophageal balloon to measure extramural pressure. In addition, capillary blood gas analysis has been recommended to ensure accurate PA catheter placement (Zone 3).

Left ventricular distention is caused by transmural pressure, which is defined as the difference between intravascular pressure

(LV pressure, measured by PA wedge pressure), and extravascular (intrapleural) pressure. Generally intrapleural pressure is assumed to be equal to atmospheric, but in the presence of PEEP it is increased. An estimate of the pleural pressure can be made by subtracting one-half the applied PEEP increment from the measured pressure (Marini, 1985). For example, if 10 cm H_2O (7.5 torr) PEEP is applied, pleural pressure would be expected to increase 3.7 torr. Corrected PA wedge pressure should therefore equal measured PA wedge pressure minus estimated pleural pressure. If the change in PA wedge pressure is greater than one-half the applied PEEP increment, which may reflect excessive alveolar pressure, catheter position in Zone 3 is questionable (Nadeau & Noble, 1986). In the presence of lung disease associated with decreased lung compliance, the pleural pressure may only increase 1/4 to 1/3 of the PEEP increment, due to decreased PEEP transmission (Marini, 1985; Nadeau & Noble, 1986). Because lung compliance is highly variable in each patient, this method will only provide a rough estimate of the actual pleural pressure.

Use of an air-filled esophageal balloon has been suggested to increase the accuracy of pleural pressure determination. However, accuracy of the esophageal-balloon pressure may be affected by position, and lung volume, therefore, its use in the clinical setting is limited (Gillespie, 1982; Marini, O'Quin, Culver, & Butler, 1982).

Analysis of pulmonary capillary wedge blood has also been recommended to increase the accuracy of PA wedge and LA pressure

Analysis of pulmonary capillary wedge blood has also been recommended to increase the accuracy of PA wedge and LA pressure relationship. While blood gas values that reflect correct pulmonary capillary wedge confirm wedging of the PA catheter, they do not ensure that the measured PA wedge pressure is truly reflective of LA pressure (Williams, Olsen, Allen, & Yergin, 1982; Morris & Chapman, 1985; Hotchkiss et al., 1986). Therefore, monitoring of the waveform tracing, and assessment of catheter placement below the left atrium by lateral chest radiographic film were still recommended to ensure PA wedge pressure accuracy.

Effects of Positioning on PA Pressure Measurement

Supine position.

Traditionally, measurement of PA and PA wedge pressures have been completed with the patient supine and flat. While this position is considered the standard, it may be poorly tolerated in patients with increased intracranial pressure or pulmonary dysfunction. Numerous studies have been completed to evaluate the effect of position on PA and PA wedge pressures (Appendix D).

Prakash, Dikshit, Forrester and Swan (1973) published the first study of the effect of position on the accuracy of PA pressure measurements in 21 patients status post MI. The backrest was elevated from 0 to 70 degrees. Mean changes were statistically, but not clinically significant: PAS [2.3 torr, ($p < 0.01$)], RA pressure [2.9 torr, ($p < 0.001$)], and PA wedge pressure [3.4 torr, ($p < 0.01$)]. However, individual variations were clinically

significant. This study highlighted the need to evaluate individual variations, not just mean data.

Woods and Mansfield (1976), studied the effect of sequential backrest elevation from supine, flat to 20, 45, and 90 degrees and dangle in ten stable cardiac outpatients. Individual changes were generally small, but some patients demonstrated a wide and clinically significant range of pressures, particularly above 45 degrees. This study reiterated the need to use a standardized reference level when altering backrest elevation. Findings from this study were confirmed in two studies by Miller and Chulay (1982) and Chulay and Miller 1984), in a total of 47 cardiac surgery patients. In contrast, Hansen (1976) in 18 ICU patients, Burrage (1979) in 30 ICU patients, Cason and Lambert (1987) in 32 critically ill patients, and Cason, Lambert, Holland, and Huntsman (1990) in 16 cardiac patients, all found large individual variations at all backrest angles up to 45 degrees. Finally, Fournier, Mensche-Dechene, Ranson-Bitker, Valladares, and Lockhart (1979) evaluated the effect of postion change from supine to sitting in eight patients with unilateral lung disease. Mean data demonstrated a clinically and statistically significant ($p < 0.025$) difference in all PA pressures in the sitting position relative to the supine position. Therefore, placement of the patient in the sitting position for PA pressure measurement was not recommended. These five studies all had methodological limitations that made evaluation of the results somewhat difficult, and may have introduced error into the results (see Appendix D).

However, all of these studies reiterate the need to evaluate individual responses to various backrest elevations.

Woods, Gross and Laurent-Bopp (1982) used repeated measures to evaluate the effect of 0-20-0 or 20-0-20 degrees backrest elevation on PA pressures in 126 ICU patients. Only four patients demonstrated changes greater than the expected fluctuation. Based on this study, reliable pressures can be obtained with the backrest elevation up to 20 degrees, however individual variations must be evaluated.

A similar repeated measures study was performed by Laulive (1982) with backrest elevation altered 0-20-45-60-0 degrees in 30 cardiac ICU patients. Individual variations were 2 torr or less, at all backrest elevations. These findings were confirmed by Clochesy, Hinshaw and Otto (1984). In this study of 17 ICU patients, mean differences were small and not significant ($p > 0.05$). However, only mean data were presented, which limited the usefulness of the study findings.

Retailliau, McGregor-Leding, and Woods (1985) evaluated the effect of altered backrest elevation from 0-30-0 degrees or 30-0-30 degrees on LA pressure. Six of 34 (18%) cardiac surgery patients demonstrated mean pressure changes greater than 4 torr. Individual variations ranged from -6 torr to +7 torr. Based on the small mean changes it appears that LA pressure can be reliably measured up to 30 degrees backrest elevation, however individual data would need to be evaluated to confirm this recommendation.

Based on these studies accurate PA and PA wedge pressures can be attained in the supine postion with a backrest elevation up to 60 degrees, and LA pressure with backrest elevation to 30 degrees as long as a standardized reference point is used. However, all of these studies have demonstrated the need to evaluate each individual's response to the backrest elevation.

Lateral position.

The issue of the effect of lateral positioning for PA pressure measurement is complicated by the fact that a standardized reference point has not been developed for each angle in the lateral position. As the body rotates, the left atrium shifts. For accurate measurement, the position of the left atrium needs to be identified. In addition, depending on the side to which the patient is placed, the PA catheter may be in a dependent or nondependent position, which affects the accuracy of the PA readings.

Nine studies were found that evaluated the effect of lateral positioning on PA pressure measurement accuracy. Murphy (1977), Bryant and Kennedy (1982), Kennedy, Bryant and Crawford (1984) and Guenther, Kay, Cheng and Lauer (1987) studied the effect of the lateral decubitus (90 degree) position on the accuracy of PA pressure measurements relative to the flat, supine position. The reference level for all these studies was the fourth intercostal space and midsternum, but position of the catheter was not specified. Murphy's study, in eight critically ill patients, found that 82% of the individual changes were less than 4 torr. The studies by Bryant and

Kennedy (1982), and Kennedy and colleagues (1984) in 25 ICU patients,
demonstrated individual changes in PA pressures of 3 torr or less.
Mean pressure differences were all 1 torr or less. Guenther and
coworkers (1987), studied 12 critically ill patients in the supine
and right and left lateral decubitus position. Mean PA wedge
pressure changes associated with the move from the supine to the
lateral position were not clinically (1 torr) or statistically (no
data given) significant. However, these findings were limited by the
lack of use of individual data. These studies indicated that
reliable PA pressures can be obtained in the 90 degree lateral
position, but individual responses to position change need to be
assessed.

Whitman (1982) evaluated the effect of a 20-degree right and
left lateral position change in 50 acutely ill cardiac surgery
patients. Mean changes were small, but individual variations were
clinically significant. Methodological limitations may have
introduced error into the results of this study.

Wild (1983) evaluated a 30-degree right and left lateral
position change in 30 critically ill patients. The intersection of
the fourth intercostal space and midsternum was used as the lateral
reference point. Clinically significant changes occurred in 28 of 30
patients, and all mean changes were statistically significant
(p < 0.01). This study is noteworthy for its' exact methodology, and
reiterated the need to determine a lateral reference point.

Keating, Boylard, Eichler and Reed (1986) studied the effect of an "approximate" 45-degree lateral turn in 20 critically ill patients with multi-system failure. The intersection of the fourth intercostal space and midsternum was used as the reference point. All lateral PA pressures were statistically ($p < 0.003$) and clinically different from the flat, supine PA pressures.

Two studies have evaluated the effect of lateral positioning with backrest elevation. Osika (1989) evaluated the effect of a 90-degree lateral turn with backrest elevation of 20, 30, and 45 degrees in 21 critically ill patients. Groom, Frish, and Elliott (1990) studied a 20-degree lateral turn with backrest elevation of 0 or 20 degrees in 30 surgical ICU and 29 medical ICU patients. Both studies found clinically significant differences in PA pressures relative to the flat, supine position. The latter study had a large number of uncontrolled variables, which prevented any recommendation based on the data.

Based on a review of these studies, the only position that has been shown to provide reliable PA and PA wedge pressures relative to the flat, supine position, is 90-degrees lateral with 0-degree backrest elevation. The reference point used in these studies was the fourth intercostal space and midsternum. All other degrees of lateral turn were associated with large individual variations. Until a specific reference point for these lateral positions is determined, and controlled studies are performed, only the 90-degree lateral

position can be recommended. As with the supine backrest elevation studies, individual responses to position change must be assessed.

Further research needs to be conducted using the fourth intercostal space, left parasternal border in the left lateral decubitus position, and the fourth intercostal space at the midsternum in the right lateral decubitus position. In addition, placement of the catheter relative to the LA needs to be described.

Complications of Hemodynamic Monitoring

The high complication rate associated with the PA catheter is frequently sited as a reason not to use the catheter. This section discusses four major complications associated with the PA catheter: 1) arrhythmias, 2) infection, 3) PA perforation, and 4) thrombosis. Incidence rates, risk factors and implications for practice will also be addressed.

Cardiac arrhythmias.

The most common complication associated with the insertion of the PA catheter is the occurrence of cardiac arrhythmias. The arrhythmias are thought to result from endocardial irritation as the catheter passes through the right ventricle. The frequency of premature ventricular complexes (PVCs) ranges from 11% (Boyd, Thomas, Gold, & Boyd, 1983) to 75% (Sprung et al., 1982). The wide variance in the frequency is related to several factors. First, Sprung, Jacobs, Caratis, and Karpf (1981) reported a PVC incidence of 48%, and a ventricular tachycardia (V tach) incidence of 33% from direct observation of the ECG screen during insertion. Permanent recordings

in these same patients demonstrated an incidence rate of 68% for PVCs

and 48% for V tach. Most of the reviews do not specify the method

for recording the ectopy; therefore, comparison among studies is

limited. The second factor is the type of arrhythmia recorded.

While the overall frequency of arrhythmias ranged from 12% to 75%,

the incidence of advanced ventricular arrnythmias, defined as greater

than three PVCs in a row (V tach), ranged from 12.5% (Johnson, 1986;

Iberti, Benjamin, Gruppi, & Raskin, 1985) to 53% (Sprung et al.,

1982). Third, the length of time required for catheter insertion is

perhaps the key variable in the wide variety of reported incidence.

In the studies conducted by Johnson (1986) and Iberti and coworkers

(1985), average insertion time in both reports was 175.9 seconds,

compared with the studies conducted by Sprung and coworkers (1981,

1982) where mean insertion time was 40 minutes (range 8 to 180

minutes) and 30 minutes (range 1 to 180 minutes) respectively. The

large variance in time difference would seem to explain the

difference between the 12% incidence rate in the Iberti and Johnson

studies and the 53% incidence in the Sprung studies.

Because of the high frequency of arrhythmias associated with PA

catheter insertion, use of prophylactic lidocaine has been

recommended. In a study by Shaw (1979) prophylactic lidocaine was

administered to 36 pre-cardiac surgery patients who served as their

own controls. Administration of lidocaine resulted in a significant

($p < 0.001$) decrease in all ectopy. In the same study normal saline

was administered to a control group ($n = 37$), and did not

signficantly alter the frequency of arrhythmias ($p > 0.05$). However, the recommendation to use lidocaine prophylactically in all patients cannot be supported. In all studies, less than 3% of the ventricular ectopy failed to resolve spontaneously or with removal of the PA catheter (Sprung et al., 1981; Iberti et al., 1985; Johnson, 1986).

In light of the low incidence of ectopy requiring definitive therapy, it is important to identify clinical risk factors most often associated with the occurrence of advanced ventricular arrhythmias. Risk factors that have identified include the presence of shock, acidosis (pH less than 7.2), hypoxemia (P_{aO2} less than 60 torr), potassium less than 3.5 mEq/L, calcium less than 8.0 mg/dL (corrected), and myocardial ischemia/infarction (Sprung et al., 1981). In studies conducted by Sprung and coworkers (1982, 1985) none of these risk factors was found to be highly specific. However, the likelihood of developing advanced cardiac arrhythmias was significantly higher ($p < 0.05$ and $p < 0.01$ respectively) in patients with both prolonged catheter insertion time (greater than 20 minutes) and any of these risk factors. Consideration of the use of lidocaine in this population may be warranted.

The other arrhythmia associated with the insertion of the PA catheter is the development of either right bundle branch block (RBBB) or complete heart block. As the catheter passes through the right ventricle it may traumatize the right bundle branch, because of its superficial location in the right ventricular endocardium. In most cases the development of RBBB is transient (Sprung, Elser,

Schein, Marcial, & Schrager, 1989) and does not require therapy.

However, in patients with preexisting left bundle branch block

(LBBB), the inducement of RBBB and subsequent complete heart block

has been reported in 0% to 22% of cases (Thompson, Dalton, Lappas, &

Lowenstein, 1979; Morris, Mulvhill, & Lew, 1987; Sprung et al.,

1989). Because of the relatively low incidence of complete heart

block developing as a result of PA catheter insertion, the presence

of LBBB is not an absolute contraindication for insertion of a PA

catheter; however, because of the potential for complete heart block

it has been recommended that a method for ventricular pacing be

immediately available for this group of patients.

Finally, during withdrawal of the catheter isolated PVCs have

been noted in 27% (Johnston, Royster, Beamer, & Prough, 1984; $N = 30$

cardiac surgery patients) to 63% (Damen, 1985; $N = 250$ cardiac

surgery patients) of patients, all were self limited. More severe

arrhythmias, multiform PVCs, were noted in 40% to 47% of cardiac

surgery patients, and V tach was not in 0.8% to 3% of patients

(Johnston et al., 1984; Damen, 1985), again, these arrhythmias

resolved spontaneously (Johnston et al., 1984). No factors were

found to predict the occurrence of these arrhythmias (Damen, 1985).

These findings emphasized the need to monitor the patient not only

during insertion but during removal of the PA catheter as well.

Infectious complications.

The overall incidence of catheter related infection varies from

0% to 33% (Elliott, Zimmer, & Clemmer, 1979; Sise et al., 1981;

Horst, Obeid, Vij, & Bivins, 1984; Shah, Rao, Laughlin, & El-Etr, 1984; Myers, Austin, & Sibbald, 1985; Samsoondar, Freeman, Coultish, & Oxley, 1985; Damen & Bolton, 1986). Patients with a previous source of infection consistently demonstrate a higher incidence of positive catheter tips ($p < 0.05$) than those without infection (Michel, Marsh, McMichan, Southorn, & Brewer, 1981). In this same study, positive catheter tip cultures were found in 21 of 153 patients; however, only one case of catheter related sepsis occurred. Therefore, the presence of a positive catheter tip culture does not necessarily indicate the presence of infection related to the catheter, but may reflect either contamination or colonization of the catheter from another source. The technique recommended to identify a positive catheter is the use of the semi-quantitative method described by Maki, Weise, and Sarafin (1977). However, numerous techniques have been used; therefore, comparison of studies is somewhat limited.

Methods recommended to decrease the incidence of infection include: 1) using aseptic technique when manipulating stopcocks and caps, 2) using single-dose heparin when preparing flushing solution (Covey, McLane, Smith, Matasic, & Holm, 1988), and 3) using normal saline instead of a dextrose solution for flush solution (Keeler, McLane, Covey, Smith, & Holm, 1987; Mermel & Maki, 1989). The presence of a sterile sleeve has not been found to decrease the rate of infection; therefore, careful handling of the catheter needs to be emphasized even though the sheath is in place (Damen & Bolton, 1986;

Keeler et al., 1987). No specific recommendations for insertion site care have been developed; however, Abbott, Walrath, and Scanlon-Trump (1983) found a nonsignificant ($p > 0.05$), but positive relation between insertion-site infection and positive catheter-tip infection; therefore, aseptic site care was recommended. Changing flushing solution every 48 hours does not appear to increase the risk of infection (Covey et al., 1988; Mermel & Maki, 1989). Results regarding the relationship between infection rate and length of insertion are equivocal, after 72 hours close observation of the patient is warranted (Elliott et al., 1979; Michel et al., 1981; Sise et al., 1981; Wunderink, Popovich, Eichenhorn, & Saravolatz, 1984; Damen & Bolton, 1986; Hudson-Civetta, Civetta, Martinez, & Hoffman, 1987). In patients with a known source of infection, consideration should be given to changing the catheter 24 to 48 hours after initiation of antibiotic therapy (Mermel & Maki, 1989) .

Pulmonary artery perforation.

The most serious complication associated with the PA catheter is PA perforation. Reports of perforation are generally anecdotal, but estimates of frequency include 0.08% (Pellegrini et al., 1987), 0.2% (McDaniel et al., 1981; Boyd et al., 1983), and 1.5% (Fraser, 1987). In the latter study the perforations were found during autopsy, and not felt to be the direct cause of death in three of four cases. Despite the relatively small occurrence, mortality related to PA perforation is approximately 50% (Hannan, Brown, & Bigman, 1984).

Factors that predispose the patient to PA perforation include pulmonary hypertension (Lemen, Jones, & Cowan, 1975; Pape et al., 1979; Barash et al., 1981; Kelly, Morris, Crawford, Espada, & Howell, 1981; Hart, Ward, Gillilian, & Browley, 1982), age (greater than 60) (Hardy, Morissette, Taillefer, & Vauclair, 1983), and use of anticoagulants (Pape et al., 1979; Barash et al., 1981; Hannan et al., 1984).

Technical factors that are associated with PA perforation include distal migration of the catheter (Chun & Ellestad, 1971; Kelly et al., 1981; Hardy et al., 1983), eccentric balloon inflation resulting in either perforation of the wall with the catheter tip, or excessive pressure from the balloon on the vessel wall (Lemen et al., 1975; Shin, Ayella, & McAslan, 1977; Barash et al., 1981), and overinflation of the balloon (Haapaniemi et al., 1979; Hardy et al., 1983).

Because the critical care nurse is responsible for the maintenance, monitoring, and attainment of measurements using the PA catheter attention to these technical factors will decrease the risk of this often fatal complication. Recommendations for prevention of PA rupture include: 1) maintaining a high index of suspicion whenever a patient presents with hemoptysis (most common presenting sign) (Golden, Pinder, Anderson, & Cheitlin, 1973), 2) continually monitoring the pressure waveform during balloon inflation, 3) inflating of the balloon only to the volume (1 to 1.5 ml) at which the pressure tracing changes from the typical PA pressure waveform to

the PA wedge pressure waveform (Swan & Ganz, 1975, 1979),

4) gradually obtaining the PA wedge pressure during inflation, if

this does not occur at the point of loss of resistance in the

syringe, the catheter may need to be pulled back (Pape et al., 1979;

McDonald & Zaidan, 1983), 5) continually monitoring the PA tracing to

detect distal migration, 6) keeping "wedge" time to a minimum, 10 to

15 seconds or three to four respiratory cycles, especially in

patients with pulmonary hypertension (Pape et al., 1979; Swan & Ganz,

1979), 7) avoiding inflation of the balloon with fluids, 8) noting

the balloon inflation volume, if "wedge" is recorded at a balloon

volume less than that on the catheter shaft, the catheter needs to be

pulled back to a position from which full or near full balloon

inflation volume produces a wedge tracing (Pape et al., 1979), and

9) following the patient with serial chest radiographic films, if the

catheter tip is greater than 5 cm from the mediastinum, the catheter

will need to be repositioned (Kelly et al., 1981).

Pulmonary artery catheter induced thrombosis.

Thrombosis associated with the PA catheter has been reported in

varying frequencies. Studies that diagnosed the presence of

thrombosis found an average occurrence of 0.9% to 2.8% (Elliott,

Zimmerman, & Clemmer, 1979; Darst & Forker, 1982; Horst et al.,

1984). However, an intraoperative study that evaluated the presence

of thrombosis in ten coronary artery bypass graft surgery patients

found thrombus development on 100% of the catheters (Hoar, Stone,

Wicks, Edie, & Scholes, 1978). Angiographic evaluation in 33

critically ill patients found an incidence of 66% (Chastre et al.,

1982). The introduction of heparin-bonded catheters was heralded as

a method of decreasing thrombus formation. Hoar and coworkers (1981)

compared heparin-coated versus non-heparin coated PA catheters

approximately 1.5 hours after insertion in 20 cardiac surgery

patients. The authors found a 100% incidence of thrombus formation

in the non-heparin group (n = 10), and a 0% incidence in the heparin-

bonded group (n = 10). Mangano (1982) studied 30 cardiac surgery

patients and found a 90% efficacy rate in preventing thrombus

formation associated with a heparin-bonded catheter, compared with a

0% efficacy rate from the non-heparin bonded catheter. However,

Mollenholt, Eriksson, and Andersson (1987) in a study of 20 ICU

patients failed to find a difference between heparin-bonded and non-

heparin bonded PA catheter's ability to prevent thrombus formation 48

hours after insertion. Therefore, the efficacy of heparin-bonding to

prevent thrombus formation is limited to a relatively short period of

time. Possible sequelae related to thrombus formation include

pulmonary embolus, septic phlebitis, and interference with PA

catheter function.

Studies by Rull, Aguirre, de la Puerta, Millan, and Maldonado

(1984) and Kim, Richman, and Marshall (1980) have identified the PA

catheter as a cause of thrombocytopenia. These authors suggested the

catheter caused an increase in consumption in platelets, secondary to

microaggregation on the catheter. Additionally, the heparin-bonded

catheter has been found to induce a thrombocytopenia syndrome, which

can only be resolved by removal of the catheter (Laster & Silver, 1988).

As noted there are risks associated with PA catheterization. However, most of the complications can be prevented by careful monitoring and assessment of risk factors. The critical care nurse is responsible for prevention of these complications, and in the case of a complication, rapid assessment and intervention.

Taxonomy of Cognitive Domains

Bloom, Englehart, Furst, Hill, and Drathwohl (1956) proposed a cognitive domain taxonomy to classify learner behaviors in response to specific instruction. The taxonomy is based on the concept of increasing complexity. The purpose of the taxonomy is not to describe how learning occurs, but to facilitate communication regarding expected behaviors. The use of this taxonomy allows one to know clearly what is meant by such terms as "understand" or "comprehend". Bloom identified six different levels in the cognitive domain. Within each level are hierarchical subsets. For each subset, behavioral objectives and evaluation criteria may be developed. This study will include all levels of the taxonomy. The levels will be combined into three cognitive groupings: Level 1, knowledge and comprehension; Level 2, application and analysis, and Level 3, synthesis and evaluation. These groupings are consistent with the test blueprint for the Critical Care Registered Nurse (CCRN) certification examination (Sullivan, Sanford, & Samph, 1988).

The most basic level of the taxonomy is knowledge (Bloom et al., 1956). This level contains six subsets, ranging from knowledge of specifics such as dates, terminology, and facts to more advanced knowledge related to how we deal with specifics, including criteria, classifications, and categories. Finally, the knowledge level addresses the more complex abstract concepts of principles and theories. At this level of the cognitive domain, the individual is required only to know about concrete and abstract phenomena discussed. Testing at this level of the cognitive domain may require the individual to recall specific bits of information. Clinically, knowledge is demonstrated by the recall of specific facts, such as the normal range for a given PA pressure measurement; a definition, such as phlebostatic axis or preload; or the ability to recall generalizations or principles, such as Starling's law of the heart; or the relationship between hydrostatic pressure and fluid flux.

The second level of the taxonomy is comprehension (Bloom et al., 1956). Comprehension refers to the understanding of the literal message contained in the communication. There are three types of behavior classified under comprehension in increasing complexity: translation, interpretation, and extrapolation of specific communication. Translation is defined as the ability to put the communication into another form of communication. Clinically, translation can be demonstrated by the ability to 1) simplify a complex, abstract technical phrase into a more concrete phrase, e.g., afterload reduction, and 2) translate symbolic forms into verbal

forms, or vice versa, e.g., reading a ventricular function curve. Interpretation may require the individual to reorder the communication and to think about the relative importance of the ideas and their interrelationships. Clinically, interpretation is demonstrated by the ability to produce inferences or summarizations within the limits of the given information. Extrapolation is the most complex behavior in the comprehension level and includes the ability to make predictions based on provided information, or to make inferences with respect to the implications or effects of given information. Clinically, extrapolation is demonstrated by the ability to predict likely effects of a given drug therapy in relation to a given set of hemodynamic parameters. In addition, extrapolation requires the ability to recognize factors that may limit the accuracy of predictions. Demonstration of comprehension requires that the individual possess the knowledge of the specific and abstract concepts. The individual is not required to utilize any of this information, but simply to demonstrate the ability to interpret, translate, or extrapolate specific information.

Application, the third level of the cognitive domain, requires knowledge of specifics and the ability to comprehend communication (Bloom et al., 1956). The difference between comprehension and application is that with comprehension the learner can use an abstraction when its use is specified; in application, the learner demonstrates the ability to use an abstraction in a situation where its use is not specified. In application, the learner is able to

specify a generalization or principle to support an action, and to relate the principles to the problem (Reilly & Oermann, 1990). This level of the taxonomy represents the ability to transfer knowledge to a unique situation. Testing of the application level requires the provision of a novel experience that requires the individual apply an abstraction in a practical manner. In devising an application level question, it is important not to provide too many clues. The goal of testing at this level is to evaluate the individual's problem-solving ability, not their ability to apply a specified abstraction. Clinically, application is demonstrated by the ability to choose a correct principle and utilize it to resolve a given problem, e.g., choice of the correct medical therapy for a given hemodynamic subset.

The fourth level of the cognitive domain is analysis. *Analysis* "emphasizes the breakdown of material into its constituent parts, and detection of the relationships of the parts, and the way they are organized" (Bloom et al., 1956, p. 144). The learner is expected to classify the elements of the communication, make explicit the relationships among the elements, and recognize the the arrangement and structure that holds the communication together. Clinically, analysis is demonstrated by the ability to recognize diagnostic cues and their relationships, and to analyze the relationships of the cues. Testing of analysis requires introduction of new material (not necessarily familiar) to the learner. The ability to use new material requires analysis, and not simply recall. The learner

should be given a scenario that requires analysis of the cues, and their interrelationships.

Synthesis is the fifth level of the cognitive domain and is defined as the pulling together of elements and parts to form a whole. Synthesis is the process of combining elements in such a way as to constitute a pattern not clearly present before. Synthesis differs from the lower levels of the taxonomy in that the learner must work with a set of elements that do not necessarily constitute a whole. The task is to draw on information from many sources, and to organize the information into a meaningful pattern. Clinically, synthesis is reflected by the creation of a specific plan of care, or a diagnostic statement. Testing of synthesis requires the provision of elements (diagnostic cues), with the learner developing a meaningful plan of care or diagnostic statement.

The highest level of the cognitive domain is evaluation, defined as the making of judgments about the value, for some purpose, of ideas, solutions, and methods. The judgment is based on the use of criteria and standards to evaluate the elements for accuracy or efficacy. Clinically, evaluation is reflected by therapeutic decision-making, e.g., selection of an appropriate intervention or therapeutic, consistent with standards of practice.

Testing of evaluation requires comparison of the response or decision for logical accuracy and consistency, with criteria or standards. Clinically, the criteria used are the standards of care. In addition, the responses are evaluated against external criteria,

i.e., expected or desired outcomes. Clinically, evaluation relative to external criteria is demonstrated by correct therapeutic decision making, i.e., actions that result in the desired or expected clinical outcomes.

Through use of the various levels of the taxonomy, it becomes clear as to what behaviors are expected from an individual and what type of evaluation is required. A logical outcome of using the cognitive taxonomy is the ability to create congruence between expected behaviors and the evaluation methods.

Simulation as an Evaluation Tool

Simulation was defined as "a method of representing one aspect or subset of reality" (Fought, personal communication, 1991). "Simulations attempt to replicate essential aspects of reality so the actual situation may be better understood and/or controlled" (Wolf & Duffy, 1979, p. 3). There are a number of formats for simulation. Of particular interest to this study is the paper-and-pencil method using a case study. The paper-and-pencil case simulation is particularly useful for assessing diagnostic and management skills (Maatsch & Gordon, 1978). Schneider (1979) indicated that paper and pencil tests were useful for evaluating all of the cognitive aspects required for nursing practice.

Two questions must be asked when evaluating critical care nurses' knowledge of PA pressure measurement. First, what does the nurse know, and second how do they utilize this knowledge? There is great debate in the nursing literature with respect to which

evaluation tool most accurately answers these questions. This controversy includes the use of simulation as an evaluation tool. There are a limited number of studies judging the effectiveness of simulation as an evaluation tool in practicing registered nurses. The studies using simulation are generally related to the evaluation of the process of decision-making, rather than demonstration of clinical proficiency in a specific area.

Del Bueno (1983) evaluated the effectiveness of simulations developed to teach and assess nurses' clinical decision-making skills. Del Bueno chose this evaluation method versus real life for several reasons: "potential risk to the patient....lack of specific opportunity and lack of required context or circumstances needed for practice" (p. 8). Del Bueno also noted that simulation allowed for the maintenance of consistency through the control of independent variables. In addition, Del Bueno noted that "video simulation proved to be a useful, reliable method to assess the ability to make specific clinical decisions" (p. 10). No statistical data were reported for this study. Without these statistical data, it was impossible to make any recommendations based on this particular tool.

DeTornyay (1968) developed a written simulation to evaluate nursing students' problem-solving abilities. Content validity was established by having nurses and a physician evaluate the simulation as a tool and by comparison with the literature. The tool was administered to nursing students and non-nursing students (no sample size was given). The difference in their scores was significant

($p < 0.001$). DeTornyay stated that determination of internal
consistency reliability was not appropriate for this tool, because
every item in this simulated clinical problem test was interrelated
with other items, and the student was not required to respond to
every item. Test-retest reliability determined at four weeks for the
efficiency index was ($r = 0.55$), and ($r = 0.41$) for the proficiency
index. The test-retest reliability results were reported to possibly
be affected by the students' review of the content on the instrument
during the month between the initial test and retest. DeTornay felt
the simulation was an effective evaluation tool, because, based on
the students' behavior on the test, the instructor should be able to
predict how the student would perform in real life, and the student
could also gain insight into their own abilities. In addition, the
tool actually measured the achievement of the learning objective
related to problem-solving, rather than simply giving the "right"
answer on a test. DeTornyay also felt the simulation was useful
because it provided the students an opportunity to practice without
endangering a patient.

Verhonick, Nichols, Glor, and McCarthy (1968) use five, two
minute taped scenarios to gain insight into the type of observations
and actions nurse practitioners and to determine why the nurses took
the actions. The scenarios were selected from 14 scenarios depicting
situations and reactions that commonly occur in clinical practice.
The five scenarios were chosen after 60 nurse practitioners and
physicians viewed them. In addition, the scenarios were shown to ten

clinical nurse specialists. The specialists were asked to record their observations and recommended actions. The specialists recorded 87% of all the relevant observations; therefore, the film sequences were felt to realistically portray a valid clinical situation. The simulations were shown to 1,576 nurses. The nurses were asked open-ended questions: 1) what did you observe? 2) what action would you like to take? and 3) what led you to take this action? The responses were coded as relevant, irrelevant, or inappropriate. The taped scenarios were used because they provided a means to present the same information to a large number of individuals. Reliability of the tool and criterion related validity were not discussed.

Grier (1976) used four written case studies to evaluate decision making in 47 registered nurses. The report decribed how specific components of the simulation were developed (polling of nurses, literature review), and the determination of face validity of the tool through review by three nurses (qualifications not specified). However, the report did not address construct or criterion validity, or the reliability of the tool. Data were presented for the decision-making component of the study, but not for tool development.

McIntyre, McDonald, Bailey, and Claus (1972) used a simulated clinical nursing test to evaluate problem solving behavior of 191 baccalaureate nursing students. The experimental group received an experimental curriculum, while the control group received the standard curriculum. This complex instrument provided for

"controlled" and "free" responses. Test-retest reliability was determined at three weeks for each scale. All scales were significantly related ($p < 0.05$ or $p < 0.001$); however, no specific data related to Pearson's Product-Moment correlation were provided. Seven of 36 of the controlled response items in scoring system I, which compared the selected answer against the quality of the response, were correlated ($r = 0.20$ to 0.27) with responses from other subsystem scores. These low correlations indicated that subsystem I was perhaps an independent scale. Section II scoring classified the selected controlled response in three categories: required, helpful but not essential, and contraindicated. Although the difference between the experimental group and the control group was not statistically significant ($p < 0.10$), the experimental group consistently selected more of the required items than the control group. Section III scoring evaluated the individuals response as demonstrating simple recall, problem solving using familiar information, or complex decision making. One-way analysis of variance was used to compare the mean selection frequency of items in the three levels of decision making for the control and experimental groups. There was no significant difference ($p < 0.10$) between the control and experimental groups. Section IV scoring evaluated the subject's selection for its maximum/minimum benefit and high/low risk dimensions. Analysis of variance indicated no significant ($p < 0.10$) differences, although the experimental group chose more high-value/low risk solutions. Section V scoring assessed the quality of

clinical judgment through the assignment of efficiency and proficiency indexes. The experimental groups' scores were significantly ($p < 0.05$) higher on the proficiency and competency indexes. The format of the instrument did not allow for split-half or odd-even techniques to evaluate internal consistency reliability. Content validity was established using current literature and critical analysis by nursing and medical experts. Criterion validity was not addressed. A major limitation of this tool was its extremely complex nature, which limited its utility.

McLaughlin and coworkers (1979), constructed a clinical simulation test for the management of essential hypertension. Test-retest reliability was evaluated using 33 subjects (11 physicians and 22 nurses) who took the test again at 60 to 90 days after initial testing. The Pearson product-moment correlation was used to assess test-retest reliability of the two scales. Reliability for the psychosocial scale was ($r = 0.67$), and ($r = 0.68$) for the pathophysiological scale. Average within subject agreement between tests was 78%. Content validity was established by inspection of all the test items and videotaped patient interviews by 12 local expert practitioners and eight nationally known expert panel members.

McLaughlin, Carr, and Delucchi (1981) reevaluated the hypertension clinical simulation (CST:HYP), and an additional chronic obstructive pulmonary disease clinical simulation (CST:COPD), to assess for reliability and validity. Test-retest reliability was

assessed at 60 to 90 days using the Pearson Product-Moment
correlation. Reliability for the psychosocial scale for the CST:HYP
was (r = .67), and (r = .68) for the pathophysiological scale
(unchanged from the previous assessment). Reliability for the
psychosocial scale of the CST:COPD was (r = .71), and (r = .88) for
the pathophysiological scale.

Content validity was estimated by comparison of the test items
with the literature. The comparison indicated all essential topics
were covered. Eighty-eight to 100% of the subjects (nurses and
physicians) indicated the case required decisions similar to real-
life; 71% to 100% thought the test allowed them to demonstrate their
actual abilities; and 75% to 100% found the test challenging.

Concurrent validity was estimated by evaluation of a panel of
nurse and physician experts. Comparison of the nurses and physicians
showed statistically significant (p < 0.05) differences on 11 of 597
items (2%) on the CST:HYP, and 3% of items on the CST:COPD. The
scores from this study were then compared with the concurrent
validity assessment of the study done in 1979 to determine the degree
of difference between the experts' ratings. The two panels differed
on only 10 of 597 items (2%) of the items for the CST:HYP and 17 of
930 items (2%) on the CST:COPD. The similarity in scores indicated
that the scores from the original panel served as valid criteria for
determining the scores of other subjects.

Construct validity was estimated by comparing the proficiency
scores of physicians and nurses with premedical and nursing students,

respectively. For both the CST:HYP and CST:COPD, the proficiency scores were significantly ($\propto$ = 0.025) higher among the physician and nurse practitioner groups compared with the students. In conclusion, this study demonstratecd that both simulations were reliable and valid tools for testing in medical and nursing education.

Baumann and Bourbonnais (1982) used a cardiac case simulation and a semi-structured interview to evalute critical care nurses' decision making in an emergency situation. The study was replicated by Thompson and Sutton (1985). The simulation was designed by Baumann and Bourbonnais, and an expert clinician reviewed the simulation for feasibility and substantive content. Following presentation of the simulation, the nurses were given one minute to determinine their nursing actions and decisions. The subjects were then asked to provide rationale for their decisions. The case study was utilized for the study because it represented a situation familiar to a majority of the subjects. No discussion of the reliability of the tool was presented.

Keihm (1985) utilized a clinical vignette to determine congruence between inference and action in 110 nurses caring for a simulated acutely-ill neurological patient. The vignette was based on a computer simulation designed by a masters prepared nurse specialist. The simulation was reviewed by seven nurse experts from the field of neuroscience. There were six items representing three decisions scored on the vignette. Each decision consisted of a question related to nursing action and a multiple-choice or open-

ended response to indicate inference. The vignette was also used by Edgers (1987) and Winston-Heath (1988) to evaluate inference and action in 409 and 31 nurses, respectively. All of the authors noted that criterion-related reliability and internal consistency were not evaluated because of the nature of simulation. All three studies demonstrated that as the complexity of the situation increased, the congruence between inference and action decreased.

Sims (1989) used a simulation to evaluate 91 critical nurses' inference and action in clinical decision-making in their care of a patient with gram-negative sepsis requiring antibiotic therapy. Content validity for the tool was evaluated by a panel of eight clinical experts. Reliability was established using test-retest by giving the same test to four graduate nursing students one week apart. Test-retest agreement was 93% between the two sets of scores. The tool consisted of multiple-choice and open-ended questions. The open-ended questions were scored based on decision rules created prior to scoring. Sims recommended that the questions in this tool be restructured so that inference (what do I deduce from the cues?) rather than knowledge (do I know what to look for?) is tested. In addition, Sims recommended the development of a generic form of the test so new categories of vignettes could be developed. Criterion-related validity was not addressed.

Only one study evaluated criterion-related validity. Holzemer, Resnick, and Slichter (1986) conducted a study of the criterion-related validity of a clinical simulation used to evaluate nurse

practitioners' care of hypertensive patients. The author compared the "Patient Management Problems" clinical simulation to chart audit and direct observation. No evidence of criterion-related validity was noted. Holzemer and coworkers questioned the utility of simulation as an evaluation tool, and suggested that simulation may be ineffective because it does not provide for the contextual nature of all problems. The use of simulation may be problematic because it does not lend itself to estimation of test-retest reliability because the students "solve" the particular problem, which makes it impossible to re-administer the test at a later time. The rules for estimation of internal consistency are also violated because in these instruments the student was not expected to answer all of the questions.

All of the tool evaluations have noted the inappropriateness of assessment of internal consistency. Only Holzemer (1986) indicated that test-retest reliability was inappropriate for simulation tools. No one tool appears to be optimal with respect to reliability or validity. However, based on the reports of the participants, simulation can be used to determine thought processes in practicing health care providers. The issues that have not been resolved with respect to the use of clinical simulation as an evaluation method include the estimation of criterion validity, and whether simulation truly evaluates an individuals' performance in the clinical setting.

Criterion Referenced Evaluation

Two major approaches to evaluation are the norm-referenced and criterion-referenced methods. Criterion-referenced evaluation measures performance against a specific criteria. Bower (1974) succinctly identified why criterion-referenced evaluation is particularly useful when she stated "if student behavior is individual and unique, then there can be no right or wrong way of performing, only safe ways" (p. 500). Bower went on to indicate that criterion-referenced evaluation was advantageous for measuring specific achievement because it allowed the learner to be evaluated based on their mastery of specified criteria.

Criterion-referenced evaluation is based on objectives derived from standards that are agreed upon and established by practice experts. The items evaluated should be limited to behaviors or areas deemed essential (mandatory, critical, reflective of minimal required competencies), that are dictated by standards of competent and safe practice (Alspach, 1982). In criterion-referenced evaluation the number of test questions is based on the importance of a given behavior. The design of the tool to evaluate critical care nurses' knowledge is based on these guidelines. The tool (Appendix F) is based on 1) a current review of the literature, and 2) the outline of material in the AACN Core Curriculum for Critical Care (1991), and 3) competencies specified for the critical care registered nurse (CCRN) certification examination. In addition, the weighting of material

with respect to cognitive levels is consistent with the CCRN certification examination.

Summary

The use of the cognitive taxonomy, the simulation-evaluation method and criterion-referenced evaluation can be linked effectively for the purposes of this study. The use of specific terminology and levels of the cognitive domain from Bloom's (1956) taxonomy allowed for the clear definition of expected behaviors, which were translated into criterion-referenced items. The paper-and-pencil simulation provides a scenario that reflects the complex interrelationships between the variables associated with PA pressure measurement. In addition, use of written simulations allows for the evaluation of an individual's mastery of this information, in light of the complexity of the interrelationships. In addition, use of the taxonomy allows for the clear identification of problem areas and strengths and the communication of these results.

Statement of Purpose

The purpose of this study was to describe critical care nurses' knowledge and ability to utilize information related to PA pressure measurement.

CHAPTER III

Methods

Critical care nurses are frequently required to care for patients undergoing pulmonary artery (PA) pressure monitoring. This study described critical care nurses' knowledge and ability to utilize information related to PA pressure monitoring.

A criterion-referenced instrument was developed by the investigator to determine critical care nurses' knowledge and ability to utilize information related to PA pressure measurement. The tool, Clinical Simulation: Pulmonary Artery Pressure Measurement (CS:PAP), was based on a clinical simulation and used multiple choice questions (Appendix G). A demographic data sheet (Appendix H) was mailed with the CS:PAP.

Study Design

A descriptive-survey design was used to describe critical care nurses' mastery of information related to PA pressure monitoring. The differences in scores on the total test and the various cognitive levels and content areas were related to the demographic variables.

Sample

A random sample from 19 geographic regions, as specified by the American Association of Critical Care Nurses (AACN), were selected by AACN from its 71,000 members. Based on the number of items in the test and statistical methods of evaluation, a minimum of 285 completed instruments were desired. In order to ensure an adequate sample size, 1000 questionnaires were mailed.

Any registered nurse who was a member of AACN at the time of the sampling was eligible for participation in the study. Licensed vocational nurses, licensed practical nurses, and paraprofessionals were excluded from the sample. In addition, only nurses practicing in the United States or serving in United States military facilities (regardless of location) were included.

Operationalization of Variables

1. Critical care nurse: any registered nurse who has practiced, for any period of time, in the critical care environment.

2. Knowledge: the knowing of facts and specific bits of information (Bloom et al., 1956).

3. Comprehension: the ability to interpret, translate, and extrapolate specific communication (Bloom et al., 1956).

4. Application: the ability to use an abstraction in a situation where its use is not specified (Bloom et al., 1956).

5. Analysis: the ability to breakdown material into its constituent parts, and detection of the relationships of the parts and the way they are organized (Bloom et al., 1956).

6. Synthesis: the process of working with elements, and combining them in such a way as to constitute a pattern not clearly there before (Bloom et al, 1956).

7. Evaluation: the ability to make judgements about the value of ideas, solutions, and methods (Bloom et al., 1956).

8. Mastery: the attainment of a predetermined level of knowledge (Waltz & et al., 1991).

9. Simulation: a controlled representation of one aspect of reality (Fought, personal communication, 1991).

10. Taxonomy: an outline of cognitive behaviors based on ever increasing complexity.

Data-Producing Instrument

There were two data-producing instruments for this study. The mailed questionnaire was selected because it allowed for cost-effective contact of a large number of individuals (Dillman, 1978). Paper and pencil simulations have been shown to be effective in evaluating large numbers of individuals (Hubbard, 1978). No questionnaire to evaluate critical care nurses' knowledge and ability to utilize information related to PA pressure measurement could be found; therefore, one was developed for this study. The final CS:PAP pilot tool included 29 items reflecting various content areas and levels of the cognitive domain, based on a review of the literature (Appendix G). In addition, the Demographic Data Sheet (Appendix H) included items related to age, gender, number of years of experience in nursing, number of years of experience in critical care, number of years since active practice in the critical care environment, basic nursing education level, highest nursing education level, additional certification, hospital size, geographic location, current position, and perceived level of experience related to PA pressure measurement was mailed with the CS:PAP tool. A cover letter explaining the purpose of the study and information for protection of human subjects was also included (Appendix I).

Clinical Simulation-Pulmonary Artery Pressure Measurement

The CS:PAP was a criterion-referenced tool. A criterion-referenced tool was used because it evaluates a subject on a set of given criteria, rather than with respect to other subjects' scores. The purpose of the criteria was to determine critical care nurses' mastery of information related to PA pressure measurement, and their ability to utilize this knowledge in a clinical scenario. Demonstration of this mastery took place in three content areas related to PA pressure measurement: 1) clinical utilization, 2) technical aspects, and 3) recognition and prevention of complications. The items in the tool were based on a review of the literature related to PA pressure measurement (see Chapter II). The AACN Core Curriculum for Critical Care Nurses (1991) was used to outline the content included in the tool (see Appendices A through E, and Chapter II for research related to the content outline of the CS:PAP). Design of the test was based on guidance from the American Nurses' Association Guide to Test Item Development (Raymond, 1986) and educational experts.

A matrix was created for the development of the test items (Appendix F). Content areas included mastery of information related to 1) clinical utilization of PA pressure measurement data, 2) technical aspects of PA pressure measurement, and 3) recognition and prevention of complications associated with PA pressure measurement. The test items were written using grouped levels of the

cognitive domain: Level 1, knowledge and comprehension, Level 2,

application and analysis, and Level 3, synthesis and evaluation.

Test results.

A pilot of the tool was conducted involving 12 critical care

nurses and eight undergraduate nursing students. The mean score for

the critical care nurses was 18.9 (67%) with a range of 16 to 21

(Appendix F). Three critical care nurses evaluated themselves as

novices with respect to PA pressure measurement; therefore, their

scores were included with the undergraduates. The mean score for the

eight undergraduates and three novices was 13.0 (46%), with a range

of 9 to 18. The mean scores for the two groups was significantly

different (t = 5.08, df = 18, p = 0.0005). The subset scores are

presented in Appendix F. Correlation between the subset scores and

the total test score was determined by ScorePak using Pearson's

Product-Moment correlation. There was a high correlation between the

total test score and each of the subset scores (clinical r = 0.79;

technical r = 0.67; complications r = 0.71; cognitive level 1

r = 0.79; cognitive level 2 r = 0.92; cognitive level 3 r = 0.70).

An example of the scoring of the test using the test matrix is

presented in Appendix F. As can be seen from the example, the

individual's knowledge deficit can be identified be evaluation of the

total score and the subset scores. This individual would benefit

from learning experiences that focused on synthesis and evaluation,

with a particular focus on complications associated with PA pressure

measurement.

Reliability.

Evaluation of the reliability of the CS:PAP was completed during a pilot study of the instrument using the test-retest method in a group of 12 critical care nurses. Test-retest allowed for determination of the stability of the measure over time. The critical care nurses completed the second test two weeks after completion of the first test. Pearson's Product-Moment correlation (r_{xy}), which is a quantitative measure of the linear relationship between two sets of scores, was used to assess test-retest reliability. The value for r_{xy} ranges from -1.00 to +1.00. A perfect positive relationship is indicated by +1.00, while a perfect negative relationship is indicated by -1.00. Test-retest reliability for the tool indicated a high correlation (r_{xy} = 0.85, $p < 0.01$) between the first and second test.

Because the tool was designed as a criterion-referenced measure, two other measures of test-retest reliability were also determined: P_o and Cohen's Kappa (K). The purpose of the test-retest procedure for a criterion-referenced tool was to determine the ability of the tool to consistently classify an individual in the same category, i.e., mastery of information, on two separate occasions.

P_o, is defined as "the proportion of observed agreements in classifications on both occasions" (Waltz et al., 1991, p. 231).

$$P_o = \sum_{k=1}^{m} P_{kk}$$

where:

m = the number of classification categories

P_{kk} = the proportion of objects or persons correctly classified

in the Kth category

Cohens's *K*appa is the proportion of persons consistently

classified in the same category on both occasions beyond that

expected by chance, i.e., P_o corrected for chance (Waltz et al.,

1991).

$$K = P_o - P_c / 1 - P_c$$

where:

P_o = proportion of observed agreements in classifications on

both occasions

P_c = the proportion of chance agreements

$$P_c = \sum_{k=1}^{m} P_k P_k$$

■ = number of classification categories

$P_k P_k$ = proportion of objects or persons assigned to

category k on each measurement occasion, respectively

Kappa is used to determine the value of K_{max}, the upper limit

value that Kappa could have with a particular distribution of

results. Kappa$_{max}$ is determined by evaluating the maximum number of

consistent test classifications, and recalculation of Kappa. The

K/K_{max} ratio provides a value that can be interpreted on a standard

scale. The upper limit for K/K_{max} is 1.00. The higher the K/K_{max}

ratio, the more reliable the instrument (Waltz et al., 1991).

Test scores for the critical care nurses and undergraduates were used to determine test-retest reliability related to classification of master/nonmaster of the test information. The ideal score for a criterion-referenced test is 100%. However, the highest score for all subjects was 21 out of 28 (75%). The combined mean score was 15.65 (55.9%) (SD +/- 3.92/14%). The mean score for the critical care nurses was 19.9. (67%) (SD +/- 1.90/6.79%). The mean score for the undergraduates and novices was 13.0 (46%) (SD +/- 3.03/10.82%). A cut score of 70%, was used to differentiate master from nonmaster of the material presented *(Po* = 0.77; *Pc* = 0.52; Kappa = 0.52; and K/K_{max} = 1.00). These data indicated that the tool possessed the ability to consistently classify the subjects relative to their ability to master the material.

Content validity.

Content validity, which evaluates how the content of each item and the test as a whole matches the objective to be measured (Waltz et al., 1991), was determined using qualified experts. The experts (based on thier expertise in clinical and educational areas) were asked to evaluate each item and assess whether it was an appropriate measure of the content domain specified in the test blue-print (Appendix F). This assessment allowed for the determination of an index of item-objective congruence (Appendix F). The index provided information regarding the evaluators' ratings as to whether a specific test item measured the intended objective (Waltz et al., 1991). The method for item-objective congruence was based on the

ratings of three content experts. The experts assign a value of +1, 0, or -1 to each item, depending of the items congruence with the items' objective. A score of +1 indicated definite congruence between the item and the content domain, a score of 0 indicated the expert was undecided, while a score of -1 indicated that the item did not reflect the content domain. The content experts were asked to evaluate each CS:PAP item for content area and cognitive-level specification. An index above 0.75 (75% item-objective congruence) was used as the cut-off score for judging the validity of each test item, i.e., any item that scored less than 0.75 was evaluated for the need to be rewritten or discarded from the test pool (Waltz et al., 1984).

The index of item-objective congruence was provided by the following formula (Waltz et al., 1991):

$$I_{ik} = \frac{(M - 1)S_k - S'_k}{2N(M-1)}$$

where:

I_{ik} = the index of item-objective congruence for item i and objective k

M = the number of objective

N = the number of content specialists

S_k = the sum of the ratings assigned to objective k

S'_k = the sum of the ratings assigned to all objectives, except objective k

Based on the index, 20 of 28 items exceeded the cut score of 0.75 for item-objective congruence related to content area. The primary area of discrepancy was the assignment of an item to the clinical versus technical content area. The remaining eight items were reevaluated for congruence with the predetermined content areas. The placement into a specified content area was based on information provided in the stem, the correct response, and the operationalized definition of each content area.

There was considerable discrepancy between the scores assigned for the cognitive levels. Only 6 of 28 items exceeded the 0.75 cut score. Twelve of the items received an index score of 0.50, and the remaining 10 scores ranged from 0.00 to 0.42. As Bloom and coworkers (1956) noted, during the initial design of the cognitive taxonomy, evaluation of cognitive behavior can only be determined by analysis of the problem presented relative to the individual learner's background of experience. Without knowledge regarding learning prior to the testing situation, exact determination of the process required to solve the problem is difficult. With this limitation in mind, the items receiving an index score of less than 0.75 were reevaluated by the investigator using the definitions operationalized in the conceptual framework. The final assignment of an item to a content area and cognitive level is outlined in Appendix F.

Content validity of the test was also estimated by the average congruency percentage. The congruency percentage is the proportion of items rated congruent by each judge (Waltz et al., 1991). The

ideal average percentage value is greater than 90%. For the
cognitive levels the average congruency was 70%. This 70% congruence
value may indicate unclear domain specification or difficulty in
evaluating cognitive level for an unknown group of subjects. The
congruency percentage for the content area was 86%. These findings
were consistent with the item-objective congruence index. Further
clarification of domain specifications may have improved the
congruency percentage.

In addition to the item-objective congruence index and
congruency percentage, two content area experts were asked to
evaluate each item for relevance to the specified content domain.
The technique used involved the use of a 4-point scale where 1 equals
not relevant, 2 equals somewhat relevant, 3 equals quite relevant,
and 4 equals very relevant (Waltz et al., 1991). The experts'
ratings were used to calculate P_o, Kappa, and a content-validity
index. In this case, P_o represents the the consistency of the
experts' ratings of the group of items within a specified content
domain. An acceptable level for P_o was greater than or equal to
0.80, and a Kappa greater than or equal to 0.25 (Waltz et al., 1991).
The initial scores received from the two experts were quite
different. Waltz and coworkers (1991) suggested two possible
problems when either P_o or Kappa were low: 1) the test items lack
homogeneity and the domain is poorly defined, or 2) the raters may
have interpreted the rating scale labels differently. One rater gave
highly relevant (4) scores to multiple content areas for the same

item. In order to clarify the discrepancy related to scoring, the directions for completion of the index were clarified, and the domain specifications were made more explicit. Following rescoring of the items by the content experts, total test P_O = 0.60, P_C = 0.42, Kappa = 0.31, Kappa$_{max}$ = 0.33, and Kappa/Kappa$_{max}$ = 0.94. However, subscores were different: 1) clinical - P_O = 0.57, P_C = 0.57, Kappa = 0.0; Kappa$_{max}$ = 0.0; 2) technical - P_O = 0.47, Pc = 0.13, Kappa = 0.13, Kappa$_{max}$ = 0.23, Kappa/Kappa$_{max}$ = 0.56; and 3) complications - Po = 0.79, Pc = 0.57, Kappa = 0.51, Kappa$_{max}$ = 0.51, Kappa/Kappa$_{max}$ = 1.00. The most likely explanation for the different in test scores was the unclear definitions and operationalization of the clinical and technical domains.

The index of content validity (CVI) was calculated based on the two content experts ratings to determine the proportion of items rate as quite/very relevant (3 or 4) by both judges. The CVI for the total test was 0.30, i.e., 30% of the items were rated as quite/very relevant by both experts. The CVI score was consistent with the item-relevance index. Again, further clarification of the clinical and technical domain specifications appears to be required.

Finally, the critical care nurses were asked to evaluate the test items for clarity. In addition, they were asked to suggest items that should not be included in the tool as well as items that should be added to the tool.

<u>Item classification</u>.

Based on the assessment of content validity, the test items were classified as follows:

Item 1. Correct answer = B; Content area = clinical; Cognitive level = 1. Knowledge of specific facts, i.e., the definition of preload.

Item 2. Correct answer = B. Content area = clinical. Cognitive level = 1. Knowledge of specific facts, i.e., the definition of afterload. The stem of this item was clarified.

Item 3. Correct answer = C. Content area = clinical. Cognitive level = 2. Analysis of elements of information, the recognition of the relationships among the elements, and the structure that holds the elements together. This item requires the recognition of the relationships between diagnostic cues, and the subsequent diagnosis of pulmonary edema.

Item 4. Correct answer = D. Content area = clinical. Cognitive level = 2. Analysis of relationships. This item requires the recognition of the relationship between PA pressures and the clinical signs of pulmonary congestion.

Item 5. Correct answer = D. Content area = clinical. Cognitive level = 3. This item requires synthesis of information presented in items 3 and 4, and the development of a goal for the treatment of pulmonary edema without systemic hypoperfusion.

Item 6. Correct answer = C. Content area = clinical. Cognitive level = 3. This item requires synthesis of information

from items 3 through 5, and specification of a therapy based on this information.

Item 7. Correct answer = C. Content area = clinical. Cognitive level = 3. This item requires synthesis of the presented information and combining the elements in a pattern not previously present. This item requires knowledge of normal variations in PA pressures and the clinical manifestations of altered preload, afterload, and contractility.

Item 8. Correct answer = B. Content area = clinical. Cognitive level = 3. This item requires synthesis of information and the development of a plan of care. The subject is required to identify the clinically important data based on knowledge of the physiologic response to diuretic therapy, and the lag time between PA pressure changes and clinical changes. Answer D was modified to make it a more appropriate distractor.

Item 9. Correct answer = C. Content area = technical. Cognitive level = 1. This item requires knowledge of specifics, i.e., the definition of phlebostatic axis.

Item 10. Correct answer = B. Content area = technical. Cognitive level = 1. This item requires translation of a symbolic form (the PA tracing) into verbal form. In addition, this item requires that the individual possess knowledge related to reading the waveform at end-expiration, determination of mean pressure, and determination of diastolic and systolic pressures. This item was expanded into four questions for the final test (items 12 to 15),

requiring correct identification of each waveform pressure, independent of other data.

Item 11. Correct answer = D. Content area = clinical. Cognitive level = 2. This item requires analysis of clinical data and recognition of the interrelationships of the cues. In the final tool this was item 16.

Item 12. Correct answer = A. Content area = clinical. Cognitive level 3. This item required evaluation of the PA waveform with criteria, with the resultant recognition of the abnormal waveform characteristic. Based on feedback from the content experts, this question was eliminated because of its difficulty and lack of importance as a criterion item.

Item 13. Correct answer = D. Content area = clinical. Cognitive level = 3. This item required synthesis of the data presented in items 10 and 11, and recognition of the clinical manifestation of this data. In the final tool this was item 17.

Item 14. Correct answer = C. Content area = clinical. Cognitive level = 3. This item requires synthesis of information presented in items 10, 11, and 13, and the development of a plan of care for the treatment of cardiogenic shock (systemic hypoperfusion and pulmonary congestion). The stem of the item was clarified to indicate the source of information required to correctly answer the item. In the final tool this was item 18.

Item 15. Correct answer = D. Content area = clinical. Cognitive level = 2. This item requires application of a therapy

based on information presented in items 10, 11, 13, and 14. Placing

of the item in level 2 assumes the subject has made the correct

clinical diagnosis and is able to specify the therapeutic principles

required in the treatment of cardiogenic shock. In the final tool

this was item 19.

Item 16. Correct answer = D. Content area = clinical.

Cognitive level = 2. This item requires application of information

based on items 10, 11, 13, 14, 15. The rationale for the cognitive

level selection is the same as item 15. In the final tool this was

item 20.

Item 17. Correct answer = D. Content area = clinical.

Cognitive level = 1. This item requires knowledge of the parameters

used to describe left ventricular end diastolic pressure. In the

final tool this was item 21.

Item 18. Correct answer = C. Content area = clinical.

Cognitive level = 1. This item requires knowledge of the parameters

used to describe cardiac work. The correct answer, choice C

(ejection fraction) was confusing because it is indirectly affected

by cardiac index. Therefore, for the final tool PA wedge pressure

was identified as the correct choice. In the final tool this was

item 22.

Item 19. Correct answer = C. Content area = clinical.

Cognitive level = 2. This item required application of principles

related to therapy effect. The stem of the question was clarified to

indicate the items that supported this question. In the final tool this was item 23.

Item 20. Correct answer = D. Content area = complications. Cognitive level = 2. This item required analysis of the data and the recognition of the relationships between the diagnostic cues, i.e., right ventricular waveform and right ventricular premature contraction indicating PA catheter placement in the right ventricle. In the final tool this was item 24.

Item 21. Correct answer = C. Content area = complication. Cognitive level = 3. This item required the synthesis of information presented in item 20, and the development of an appropriate plan of care. In the final tool this was item 25.

Item 22. Correct answer = D. Content area = technical. Cognitive level = 2. This item required analysis of the dynamic pressure waveform and application of principles related to technical monitoring of the pressure system. This item was omitted from the final tool, because of lack of importance as a criterion item.

Item 23. Correct answer = C. Content area = technical. Cognitive level = 2. This item required analysis of the effect of positive end-expiratory pressure (PEEP) on the PA wedge-left atrial pressure relationship. This item was modified for the final, and required the subject correctly calculate the effect of a specified PEEP on the PA wedge pressure. In the final tool this was item 26.

Item 24. Correct answer = C. Content area = technical. Cognitive level = 3. This item required synthesis of the information

presented and the development of an interpretive statement. In the final tool this was item 27.

Item 25. Correct answer = C. Content area = technical. Cognitive level = 2. This item required the analysis of the data presented in item 24, and the recognition of the relationship between position and accuracy of PA pressure readings. Answer D was further clarified to improve on its inaccuracy. In the final tool this was item 28.

Item 26. Correct answer = B. Content area = complication. Cognitive level = 1. This item required translation of the symbolic form (PA pressure tracing) into its verbal form. This item required knowledge related to the correct waveform characteristic, and the implication of the a permanent PA wedge pressure tracing. Answer A was also a correct answer for this item, and it was altered to provide a more appropriate distractor. In the final tool this was item 10.

Item 27. Correct answer = B. Content area = complication. Cognitive level = 2. This item, based on item 26, required application of principles related to the treatment of a permanently wedged PA catheter. Assignment of this item to cognitive level 2 is based on the assumption that the subject has correctly identified the waveform in item 26, and is able to decide which option is correct based on the scenario. Answer A was also considered a possible therapeutic option, and was clarified to make it an appropriate distractor. In the final tool this was item 11.

Item 28.   Correct answer = C.   Content area = technical.
Cognitive level = 3.   This item required synthesis of information
related to PA pressure measurement and the development of an plan of
nursing care.   In the final tool this was item 29.

The final tool consisted of 29 items (Appendix G).   The order
of the items was altered to improve flow of the material.   The final
tool was reviewed by a content expert for face and content validity.
The test blueprint and answer sheet for the final test were altered
appropriately (Appendix G).

Internal consistency.

Internal consistency was not evaluated for the CS:PAP.   The
items in the tool were designed to evaluate thought process related
to a given content area; therefore, if an individual made an
incorrect diagnosis they were also likely to provide incorrect
rationale and nursing care for a given scenario.   In addition, the
purpose of evaluating iternal consistency is to determine the degree
that all of the test items pull together.   Because the CS:PAP was
designed to test a wide variety of content, it was expected that the
tool as a whole would have low internal consistency.

Construct validity.

Construct validity, which established the tool's ability to
function in accordance with its purpose, i.e., measurement of
critical care nurses' cognitive abilities related to PA pressure
measurement, was evaluated using a contrasted group approach.   The
two contrasted groups consisted of 11 critical care nurses and a

group of eight undergraduate nursing students (individuals who had not practiced in critical care or worked with PA pressure measurement) and three graduate students who evaluated themselves as novices with respect to PA pressure measurement. The use of the contrasted group approach allowed for comparison of the two groups for the possession of the desired attribute, i.e., knowledge and the ability to utilize information related to PA pressure measurement. It was hypothesized that the critical care nurses would possess a higher degree of this attribute; therefore, their scores would be significantly higher ($p < 0.05$) than the scores of the inexperienced nurses. In addition, data from these two groups were used to determine item discrimination and item difficulty.

A one-tailed, pooled t test was used to determine if the difference between the instructed-uninstructed groups was significant ($p < 0.01$). The null hypothesis was that the mean score of the instructed group was equal to the mean score of the uninstructed group (H_o: $\bar{x}_1 = \bar{x}_2$; H_a: $\bar{x}_1 > \bar{x}_2$). The mean score for the instructed group (critical care nurses except those identified as novice) was 18.9 out of 28, and the uninstructed group (undergraduates and novice critical care nurses) was 13.0. As noted, the difference between the two scores was statistically significant ($p = 0.005$); therefore, the null hypothesis was rejected and the alternative hypothesis was supported (Appendix F).

Item difficulty reflected the percentage of respondents correctly answering a given item (Jenkins & Michael, 1986). The item

difficulty value ranges from 0.00 to 1.00 and reflects the proportion of subjects who answer the question correctly. Because the critical care nurses were expected to have a greater familiarity with PA pressure measurement, they were expected to answer more questions correctly, as demonstrated by a higher p level, e.g., lower difficulty, than the student nurse group. The difference between test scores for the critical care nurses compared with the undergraduate and novice nurses was evaluated using Chi square analysis. Based on the distribution of test scores, only 7 of 28 items were significantly different ($p < 0.05$).

In addition, item difficulty was determined for the whole sample using ScorePak Item Analysis from the Educational Assessment Center at the University of Washington. The item difficulty index for the whole sample ranged from 0 to 100; the higher the value the easier the question. To maximize item discrimination the desired difficulty level for a four-response multiple-choice item was 74 (Lord, 1952). The data related to item difficulty is outlined in Appendix F. ScorePak arbitrarily classifies items as "easy" if the index is 85% or above; moderate if it is between 51% and 84%; and "hard" if it is 50% or below. For the total sample three items were classified as "easy", 16 items were classified as "medium", and nine items were classified as "hard".

The item-difficulty index findings were consistent with the classification of test items as "medium" or "hard". Because of the "difficult" nature of the test both groups missed a large number of

the items. Two options need to be considered based on these data:

1) rewriting the items o make them "easier", or 2) acknowledgement that the items adequately addressed critical content, and should be left in the tool. Based on the evaluation of the tool, it was decided to opt for the latter option.

Item-discrimination (Appendix F) for the sample was calculated using ScorePak statistical analysis. This method of analysis provided a more accurate assessment of the discrimination power of an item because it took into account the responses of all students, not just high and low scores, although in this case the critical care nurses scored higher than the undergraduate and novice nurses (Appendix F). A Pearson Product-Moment correlation between subject responses on a particular item and the total score on all items on the test was calculated ("Item Analysis", 1989). Because the items on the CS:PAP covered a wide range of content areas, it was expected that the correlation coefficient would be lower than for a more homogenous test. ScorePak classifies item discrimination as "good" if the index is above 0.30; "fair" if it is between 0.10 and 0.30; and "poor" if it is below 0.10. Nine items were classified as "good", 13 items were classified as "fair", and six items were classified as "poor". There are several reasons why an item may have "poor" discrimination power: 1) easy or very difficult items will have low discrimination scores, but they are necessary to cover important content, and 2) a test item may show low discrimination if the test measures many different content areas and cognitive skills

("Item Analysis", 1989). The CS:PAP contained items that were considered critical content, and it was expected that all subjects would answer the item correctly. In addition, the CS:PAP contained items requiring a high cognitive skill level that were intended to discriminate between individuals who were classified as master and nonmaster of the material being tested. Finally, the purpose of the CS:PAP was to test a wide content area on different cognitive levels.

Methods of Procedure

One thousand subjects were randomly chosen by AACN from the current members of AACN who met the inclusion criteria. The mailing labels were sent to a Seattle mailing house. The subjects were asked to complete a 29 item test (CS:PAP), and a demographic data sheet requesting information related to age, gender, experience in nursing, experience in critical care, hospital size, area of employment, basic and advanced nursing education, additional professional certification, and perceived level of knowledge related to PA pressure measurement. The CS:PAP tool (Appendix G), a mark sense answer sheet, and the demographic data sheet (Appendix H) were mailed with a cover letter (Appendix I) explaining the purpose of the research, and protection of human subjects. Respondents were asked to return the questionnaire within 10 days of receipt. A self-addressed stamped envelope was provided to facilitate return of the data collecting tools. A follow-up postcard (Appendix J) was mailed to all recipients ten days after the tool was mailed, thanking those who completed the tools, and encouraging those who had not to do so.

To ensure the test only reflected current knowledge, the subjects were asked not to use any references to complete the CS:PAP tool. All participants were offered an answer sheet and an abstract of the results. The subjects were asked to complete a form attached to the tool with their name and address requesting the results of the study. To ensure anonymity, the personal information was separated from the CS:PAP mark sense score sheet and the demographic data sheet prior to scoring.

Protection of Human Subjects

Application for permission to conduct this study was made to the University of Washington Human Subjects Review Committee (Appendix M). The cover letter (Appendix I) indicated that participation in the study was voluntary, and no retribution would occur for refusal to participate in or withdrawal from the study. The 1,000 subjects were chosen randomly by the American Association of Critical Care Nurses from the AACN membership. The mailing labels were sent to a Seattle mailing house. The demographic survey and mark sense answer sheet were number coded prior to forwarding to the mailing house, so the investigator did not have a record of the members to whom the study was sent; therefore, there was no risk of professional consequence as a result of the study scores. In addition, to ensure anonymity, the subjects were asked to not place their name on any of the forms, except the request for the test answers and abstract. The latter form was separated from the demographic data sheet and mark sense answer sheet prior to scoring.

Return of the demographic data sheet and mark sense answer sheet indicated consent to participate in the study. The demographic data sheet and mark sense answer sheet will be kept indefinitely in a locked file cabinet; available only to study personnel. The results of this study may be used for thesis development, professional publication, and professional meetings. The subject will have access to the final report of this project through the University of Washington.

Method of Data Analysis

The data obtained from the CS:PAP and the DDS were evaluated using descriptive statistics. Descriptive statistics were appropriate for use in this study as they described the data through frequency distributions, central tendency and variability (Volicer, 1984). In general the analysis will proceeded in four phases:

1. Demographic data analysis: the data were analyzed for central tendency (mode, median), shape of distribution (kurtosis and skewness), and measure of dispersion (range). The statistics used for the analysis were consistent with the level of data collected, and the purpose of the study (Waltz, 1981).

2. The sample demographic data were compared with the demographic data from the AACN population using Chi square analysis or a one-tailed t-test.. Calculation of these statistics facilitated the determination of any significant differences between the sample and the target population. (Grant-Knapp, 1985).

3. From the matrix (Appendix G) six subscores and a total score, were determined. These data were analyzed for central tendency (mean score), shape of distribution (kurtosis and skewness), and variability (range and standard deviation).

4. A one-way analysis of variance (ANOVA) was used to determine if there were differences in test scores based on the specified demographic characteristics. If a significant difference ($p < 0.05$) between the means was discovered, the Tukey B, a *post-hoc* multiple comparison test was conducted to determine which demographic characteristics were different relative to total and subset test scores. When cell sizes were unequal, which was anticipated for this study, smaller groups have an abnormally high percentage of Type I errors (Petrinovich & Hardyck, 1969, Wike, 1971). In order to avoid Type I errors, if the ANOVA failed to detect a difference between the means, any statistically significant differences that were identified by the Tukey B were disregarded (Petrinovic & Hardyck, 1969, Wike, 1971). When the sample size was equal to 15 or more per cell the Tukey B was an appropriate test with regard to avoidance of Type II errors.

CHAPTER IV

Results

To describe critical care nurses' knowledge and ability to
utilize information related to pulmonary artery (PA) pressure
measurement, a questionnaire packet containing a cover letter,
demographic data sheet, questionnaire (Clinical Simulation: Pulmonary
Artery Pressure Measurement), mark-sense answer sheet, and a postage-
paid envelope were sent to a random sample of 1000 current members of
the American Association of Critical Care Nurses (AACN). A total of
181 (18.1%) responses were received within six weeks of the mailing
of the questionnaire packet. Four individuals returned the mark
sense data sheet but did not return the demographic data sheet. Item
analysis was conducted on 181 mark sense sheets, and correlation of
the demographic data with the test scores was conducted on 177
(17.7%) data sets. The use of AACN membership does not imply AACN
review or endorsement of this study.

This chapter presents the demographic characteristics of the
sample, an analysis of individual test items, total and subset
scores, and correlation of the demographic variables with the total
and subset scores. The chapter concludes with a summary of the
findings of this study.

Characteristics of the Sample

Demographic characteristics of the sample are presented in
Table 5. Analysis of the sample characteristics was based on the

Table 5.

Summary of Demographic Characteristics of the Sample

Demographic	Frequency	Sample ($N = 177$)[a] Valid Percentage[b]	AACN Valid Percentage[b]
Gender			
Male	10	5.6	----
Female	165	93.2	----
Missing	2	----	----
Age			
20-29	35	19.9	26.83
30-34	47	26.7	27.79
35-39	48	27.3	21.52
40-44	30	17.0	12.79
45+	16	9.1	11.07
Missing	1	----	-----
State of Residence			
CT,ME,MA,NH,RI,VT	13	7.47	5.72
NY	10	5.75	6.67
DE,NJ,PA.	24	13.79	10.61
DC,MD,VA,WV	11	6.32	5.94
NC,SC	5	2.87	3.76
AL,GA	8	4.60	3.23
FL,PR,GU	11	6.32	5.85
MI,WI	12	6.90	5.27
IN,OH	12	6.90	6.22
IL	6	3.45	4.89
KY,TN	6	3.45	3.07
AR,LA,MS	5	2.87	2.91
IA,MN,NE	4	2.30	2.92
KS,MO	7	2.30	3.39
OK,TX	13	7.47	7.11
MT,ND,SD,WY	2	1.15	1.01
AS,CO,NV,NM,UT	9	5.17	4.48
AK,ID,HI,OR,WA	5	2.87	4.37
CA	11	6.32	12.58
No response	3	-----	-----
Basic Education in Nursing			
Diploma	59	33.50	28.55
Associate	39	23.20	27.32
Baccalaureate or Higher	77	43.80	43.47
No Response	1	-----	-----

Table 5 (continued)

Highest Degree in Nursing			
Diploma	35	20.1	19.83
Associate	30	17.2	22.32
Baccalaureate	79	45.4	46.79
Masters or Doctorate	30	17.2	11.06
No response	3	----	-----
Certification			
ACLS Provider	125	70.6	-----
ACLS Instructor	31	17.5	-----
ACLS Affiliate	5	2.8	-----
CCRN	100	56.5	-----
Other	31	17.5	-----
Years of Nursing Experience			
Less than 3	7	4.00	12.64
4 to 5	18	10.30	15.98
6 to 10	53	30.30	29.09
11 to 15	48	27.40	24.18
16 to 20	26	14.90	12.78
21 plus	23	13.10	9.35
No response	2	-----	-----
Years of Critical Care Experience			
Less than 3	21	11.90	27.31
4 to 5	25	14.20	15.98
6 to 10	62	35.20	29.68
11 to 15	46	26.10	17.64
16 to 20	16	9.1	7.30
21 plus	6	3.4	2.09
No response	1	-----	-----
Years Since Active in Critical Care			
Less than 2	36	20.3	-----
Greater than 2	16	9.12	-----
Not applicable	122	70.12	-----
Employed in Nursing			
Full Time	131	74.00	68.17
Part Time	42	23.70	16.03
Other	4	2.3	1.40

Table 5 (continued)

Type of Facility

Community	101	57.10	61.70
University	49	27.70	19.97
Other	27	15.30	18.32

Hospital Size

Less than 199	27	15.30	16.41
200 to 299	44	24.90	18.37
300 to 399	39	22.00	18.67
400 plus	58	32.80	46.54

Area of Employment

Combined ICU/CCU	49	27.70	25.73
ICU	17	9.60	11.91
CCU	19	10.70	11.12
Surgical ICU	13	7.30	7.81
Cardiovascular Surgical ICU	19	10.70	7.58
Rotate ICU	13	4.00	-----
Other	58	33.90	36.00

Position

Staff/General Duty	93	52.50	51.05
Charge/Team Leader	20	11.30	16.27
Head/Asst Head Nurse	16	9.00	10.69
Inservice/ Staff Development	11	6.20	3.33
CNS/Nurse Specialist	16	9.00	4.47
Academic Instructor	6	3.40	2.24
Other	15	8.50	11.94

Knowledge

Novice	10	5.60	-----
Advanced Beginner	15	8.50	-----
Competent	79	44.60	-----
Proficient	60	33.90	-----
Expert	11	6.2	-----
No Response	2	-----	-----

Note (--) Missing or not applicable
a = Four subjects did not include the demographic data sheet
b = valid percent is calculated on the available data. Missing scores are not reflected in this frequency percentage.
ICU: intensive care unit; CCU: coronary care unit; CNS: clinical nurse specialist; ACLS: advanced cardiac life support; CCRN: critical care registered nurse.

demographic data sheet information completed by 177 of the

respondents. Four respondents did not return the demographic data

sheet with the mark sense answer sheet. Valid percentages (based on

available data, omitting the missing data) were used to describe the

sample. Information regarding the AACN population was based on data

provided by AACN from their "Master File Demographics: Profile

Count All Members". Chi-squared (X^2) test of proportions was used to

determine whether the differences between the observed frequencies

(sample) and the expected frequencies (AACN population) were due to

chance variation or whether there were true differences in population

proportions. Demographic characteristics included gender, age,

region (based on 19 geographic regions classified by AACN); basic

education in nursing; highest degree in nursing; certification

including advanced cardiac life support (ACLS) critical care

registered nurse (CCRN), and any other professional certification;

years of nursing experience; years of critical care experience; years

since active practice in critical care; employment status; type of

facility; size of facility; primary area of employment; position; and

perceived knowledge level related to PA pressure measurement.

Gender

The sample was composed of 165 (93.2%) females and 10 (5.6%)

males. Because of the small male sample, no statistical evaluation

of these data was conducted. In addition, AACN did not provide

information related to gender of the population.

Age

Age ranged from 24 to 67 years. Age was reclassified for comparison with the AACN population. Individuals from age group 20 to 24 (n = 2, 1.1%) and 25 to 29 (n = 33, 18.8%) were combined for analysis with the AACN population (26.83%). There was a larger number of individuals in the sample in the 35 to 39 group (n = 48, 27.3%) and in the 40 to 44 group (n = 30, 17.0%) compared with the AACN population 21.52% and 12.79% respectively. There was no significant difference (X^2 = 5.12, df = 4, p > 0.05) between the sample and the AACN population.

Region

State of residency was classified into 19 regions specified by AACN. There was no significant difference (X^2 = 7.75, df = 18, $X^2_{.95}$ = 28.869, p > 0.05) in the distribution of the sample compared with the AACN population. Data were missing on three subjects.

Basic and Highest Education in Nursing

The majority of the sample (n = 77, 43.8%) reported basic nursing education at the baccalaureate level. There was no significant difference (X^2 = 1.48, df = 3, $X^2_{.95}$ = 7.815, p > 0.05) between the sample and the AACN population. The majority of the sample (n = 79, 45.4%) reported the baccalaureate level as the highest degree in nursing. There were 30 individuals in the sample who reported a masters degree as the highest educational level (17.2%) compared with 11.06% of the AACN population. No individuals reported their highest education at the doctoral level. There was no

statistically significant difference (X^2= 5.07, df = 3,

$X^2_{.90}$ = 6.251) between the sample and the AACN population.

Certification

The majority of the sample (n = 125, 70.6%) were ACLS

providers, and had completed CCRN certification (n = 100, 56.5%).

Other certifications (n = 31, 17.5%) included basic life support

instructor, certified emergency nurse, pediatric advanced life

support, trauma provider, emergency medical technician, and American

Nurses Association clinical nurse specialist. No data were available

for the AACN population.

Years of Nursing and Critical Care Experience

There were fewer nurses in the sample with less than three

years of experience (n = 7, 4.0%) than in the AACN population

(12.64%). The largest number of subjects in the sample were in the

groups with six to ten (n = 53, 30.3%) and 11 to 15 (n = 48, 27.4%)

years of nursing experience. There was no significant difference

(X^2 = 1.48, df = 3, $X^2_{.90}$ = 6.251,) between the sample and the AACN

population. The largest number of subjects (n = 62, 35.2%) were in

the six to ten years of critical care experience classification.

There were fewer nurses in the sample with less than three years of

critical care experience (n = 21, 11.9%) compared with the AACN

population (27.31%), and there were a greater percentage of nurses in

the sample in the 11 to 15 (n = 46, 26.1%), 16 to 20

(n = 16, 9.1%), and 21 plus (n = 6, 3.4%) year groups compared with

the AACN population. There was a trend toward a significant

difference (x^2 = 10.82, df = 5, $x^2_{.90}$ = 9.236, *0.05 < p < 0.10*)

between the sample and the AACN population. Twenty percent of the

sample reported that it had been less than two years since active

practice in critical care. It was felt that many of these responses

were due to misinterpretation of the question on the demographic

sheet. One hundred and twenty two (70%) subjects reported they were

currently active in critical care. No information was provided by

AACN about status in critical care.

Employment

The majority of the sample (*n* = 131, 74.0%) reported full-time

employment in nursing compared with the AACN population (68.17%). In

addition, there was a larger percentage of nurses in the sample

(*n* = 42, 23.7%).who reported part-time employment compared with the

AACN population (16.03%). There was a signficant difference

(x^2 = 9.75, df = 2, $x^2_{.99}$ = 9.210, *p* < 0.01) between the sample and

the AACN population.

Hospital Type and Size

The majority of the sample were employed in either community

hospitals (*n* = 101, 57.1%) or university affiliated hospitals

(*n* = 49, 27.7%). There was no significant difference (x^2 = 3.83,

df = 2, $x^2_{.90}$ = 4.605) between the sample and the AACN population.

The largest percentage in the sample reported working in hospitals

with 400 plus beds (*n* = 58, 32.8%); however, this was markedly less

than the AACN population (46.54%). There was a trend toward a

significant difference (X^2 = 7.05, df = 3, $X^2_{.90}$ = 6.251,

0.05 < p < 0.10) between the sample and the AACN population.

Area of Employment and Position

The largest percentage of the sample (n = 49, 27.7%) reported primary employment in a combined intensive/coronary care unit. There was no significant difference (X^2 = 2.03, df = 5, $X^2_{.90}$ = 9.236) between the sample and the AACN population. A majority of sample reported working as staff or general duty (n = 93, 52.5%). A larger number of individuals reported primary employment as staff development or inservice coordinators (n = 11, 6.2%), and clinical nurse specialists (n =16, 9.0%) compared with the AACN population (3.33% and 4.47% respectively). There was a trend toward a significant difference (X^2 = 10.48, df = 5, $X^2_{.90}$ = 9.236, 0.05 < p < 0.10) between the sample and the AACN population.

Perceived Knowledge of Pulmonary Artery Pressure Measurement

Ten (5.6%) individuals reported their perceived knowledge level of PA pressure measurement as novice. The largest percentage of individuals (n = 79, 44.6%) reported there perceived knowledge level as competent, while 60 (33.9%) reported their perceived knowledge level as proficient. Eleven individuals (6.2%) rated themselves as expert related to PA pressure measurement.

Analysis of Test Scores

The data-generating instrument for this study was a 29-item, self-administered, criterion-referenced questionnaire (Appendix G). Questions were multiple choice, with four possible options. A total

of 69 (0.33%) items were not answered. These were interpreted as
indicative of a subject's lack of knowledge regarding the item and
were, therefore scored as wrong.

Total and Subset Scores

There were seven scores calculated for each questionnaire
(Table 6). The total score for the questionnaire, based on 29
possible, ranged from 5 to 28. The mean was 18.98 (65%), with one
standard deviation of 4.11. The median score was 19.00 (66%), and
the modal score was 20.00 (69%). The skewness was -0.257 (negative
skew), indicating a slightly higher number of scores above the mean.
The kurtosis was 0.253, indicating a slightly peaked (leptokurtic)
curve. A leptokurtic curve is characterized by less variance in
scores than a normal curve. One hundred and thirty five (72.92%) of
subjects fell within one standard deviation, 174 (96%) fell within
two standard deviations, and 180 (99.5%) fell within three standard
deviations of the mean. Only 27.07% (n = 49) of the subjects scored
above 70% (20.3 out of 29). Table 7 presents frequencies,
percentages, and a histogram of the frequency distribution for total
test scores.

In addition to the total test score, six subset scores were
calculated (Table 6). Each test item was classified into two
categories, a content area and a cognitive level (Table G-2). Each
subset score was based on the sum of the items in each subset. The
clinical subset consisted of 16 items. The mean was 12.09,

Table 6.

Group Data: Total and Subset Scores

Score Name	Total Score	Mean Score	Median Score	Mode Score	One Std Dev	Range	Skewness	Kurtosis
Total	29.00	18.98	19.00	20.00	4.11	5-28	-0.257	0.253
Clinical	19.00	12.09	12.00	13.00	2.62	5-16	-0.379	-0.387
Technical	9.00	4.28	4.00	5.00	1.86	0-8	-0.034	-0.558
Complications	4.00	2.61	3.00	4.00	1.20	0-4	-0.511	-0.587
Level 1	10.00	5.82	6.00	5.00	1.97	1-10	-0.390	0.181
Level 2	11.00	7.79	7.00	8.00	1.75	2-11	-1.200	0.181
Level 3	8.00	5.36	6.00	6.00	1.60	1-8	-0.391	-0.316

Note.   Std Dev = standard deviation; Level 1 = cognitive level 1 - knowledge and comprehension; Level 2 = cognitive level 2 - application and analysis; Level 3 = cognitive level 3 - synthesis and evaluation.

Table 7.

Summary of Total Test Score Frequencies

Score	Freq	Pct	Cum Pct	Bargraph of Relative Frequencies (Pct)
5.00	1	1	1	##
8.00	2	1	2	##
10.00	2	1	2	##
11.00	1	1	3	##
12.00	3	2	4	####
13.00	10	6	10	############
14.00	7	4	14	########
15.00	11	6	20	############
16.00	12	7	27	##############
17.00	16	9	35	##################
18.00	13	7	43	##############
19.00	18	10	52	####################
20.00	25	14	66	##############################
21.00	12	7	73	##############
22.00	13	7	80	##############
23.00	12	7	87	##############
24.00	8	4	91	########
25.00	7	4	95	########
26.00	3	2	97	####
27.00	3	2	98	####
28.00	3	2	100	####

Note. ## = 1%; or 1.81 subjects

Freq = frequency; cum = cumulative; pct = percent

with a range of 5 to 16. The Technical subset consisted of nine
items. The mean was 4.28, with a range of zero to eight correct.
The Complications subset consisted of four items. The mean was 2.61,
with a range of zero to four. Cognitive Level 1 (knowledge and
comprehension) consisted of ten items. The mean was 5.82, with a
range of one to ten correct. Cognitive Level 2 (application and
analysis) consisted of 11 items. The mean score was 7.79, with a
range of 2 to 11. Cognitive Level 3 (synthesis and evaluation)
consisted of eight items. The mean was 5.3, with a range of one to
eight. There was a statistically significant ($p < 0.05$) correlation
between four of six subset scores (clinical, $r = 0.82$; cognitive
level 1, $r = 0.76$; cognitive level 2, $r = 0.79$; and cognitive level
3, $r = 0.76$) and the total score (Table G-3). The correlation
between the score on the technical subset and the total score was
high ($r = 0.71$, $p > 0.05$), but did not reach statistical
significance. There was moderate correlation ($r = 0.53$, $p > 0.05$)
between the scores on the complication subset and the total test.

Analysis of Individual Test Items

Item analysis for individual items was completed using ScorePak
from the Educational Testing Service at the University of Washington.
Item analysis included a frequency distribution for all possible
responses, and mean scores for the individuals who selected each item
option. From these data, item difficulty and item discrimination
were determined (Table G-4). Item difficulty is the percentage of
subjects who answered the item correctly and ranges from 1 to 100.

The higher the value the easier the question. ScorePak classifies items as "easy" if the index is 85 or greater, "medium" if the index is 51 to 84, and "hard" if the index is 50 or below. For multiple choice questions with four possible responses, the ideal difficulty is 74 (Lord, 1952). Five items were classified as "easy", 19 items were classified "medium", and five items were classified as "hard".

Item discrimination refers to the ability of an item to differentiate among subjects on the basis of how well they know the material being tested. The item discrimination index provided by ScorePak is a Pearson Product-Moment correlation between subject responses on a particular item and total scores on all other items. The item discrimination index will seldom exceed $r = 0.50$. ScorePak classifies item discrimination as "good" if the index is above 0.30, "fair" if it is between 0.10 and 0.30, and "poor" if it is below 0.10. Item analysis data are presented in Table G-4. One item was classified as a "poor" discriminator, 22 items were classified as "fair", and six items were classified as "good".

Item 1 tested the subjects' knowledge of the definition of preload. The correct answer "end-diastolic volume" was selected by 85.6% (n = 155) of the sample. The distractor "end-systolic volume" was selected by 8.3% ($n = 15$), and "resistance to ejection" was selected by 5.0% ($n = 9$) of the subjects. The item difficulty index was 86 (easy), and the item discrimination index was 0.17 (fair).

Item 2 tested the subjects' knowledge of the definition of afterload. The correct answer was selected by 74.6% ($n = 135$) of the

subjects. However, 12.7% (*n* = 23) subjects chose "contractility", and 6.1% (*n* = 11) of the subjects each chose "preload" and "ejection fraction" as a measure of vascular tone. One individual did not respond to this item. The item difficulty index was 75 (medium), and the discrimination index was 0.17 (fair).

Item 3 required the subjects to analyze and recognize the relationships between clinical data that were diagnostic of pulmonary edema. The correct answer was selected by 94.5% (*n* = 171) of the subjects. Five percent (*n* = 9) subjects selected the distractor impaired systemic perfusion as the correct diagnosis. The item difficulty index was 94 (easy), and the discrimination index was 0.20 (fair).

Item 4 required recognition of the relationship between PA pressures and the clinical signs of pulmonary congestion as the basis for the diagnosis made in item 3. The correct answer was selected by 95.6% (*n* = 173) of the subjects. One subject did not respond to this item. The item difficulty index was 96 (easy), and the discrimination index was 0.13 (fair).

Item 5 required the subjects to synthesize information presented in items 3 and 4 and to identify a goal for the treatment of pulmonary edema without systemic hypoperfusion. The correct answer "decrease in preload" was selected by 66.9% (*n* = 121) of the subjects. However, 17.1% (*n* = 31) chose the distractor "increase contractility", and 14.4% (*n* = 26) chose "decrease afterload" as the

correct goal for therapy. The item difficulty index was 67 (medium), and the discrimination index was 0.20 (fair).

Item 6 required synthesis of information from items 3 through 5 and specification of therapy for a patient with pulmonary edema without systemic hypoperfusion. The correct answer "volume reduction" was selected by 70.7% (n = 128) of the subjects. The distractor "inotropic therapy" was selected by 24.9% (n = 45) of the subjects. The item difficulty index was 71 (medium), and the discrimination index was 0.20 (fair).

Item 7 required the subject to determine, by exception, if the changes in hemodynamic data were representative of a normal variation in PA pressures, or indicative of an alteration in preload, afterload, or contractility. The correct answer (exception) was selected by 65.7% (n = 119) of the subjects. However, 16.0% (n = 29) of the subjects did not recognize the hemodynamic changes as indicative of "normal fluctuation", while 9.4% (n = 17) chose "increased contractility", and 8.3% (n = 15) selected "decreased afterload". One subject did not respond to this item. The item difficulty index was 66 (medium), and item discrimination was 0.27 (fair).

Item 8 required the subjects to recognize clinically important data based on a knowledge of the physiologic effect of diuretic therapy and the lag time between PA pressure changes and clinical changes. The correct response "hold the diuretic" was selected by 81.2% (n = 147) of the subjects, while 13.8% (n = 25) chose to give

the diuretic because the patient still had clinical signs of pulmonary congestion. The item difficulty index was 81 (medium), and the discrimination index was 0.14 (fair).

Item 9 required the subject to define the term "phlebostatic axis". Only 21.5% (n = 39) of the subjects correctly defined the term. The distractor "intersection of an axis transsecting the fourth intercostal space at the sternum and the midaxillary line" was selected by 76.8% (n = 139) of the subjects. The question difficulty index was 22 (hard) and the discrimination index was 0.05 (poor). The poor item discrimination index indicated that both individuals who scored high and low missed this item.

Item 10 required the subject to correctly identify the waveform as indicative of a spontaneous wedge. The correct response was selected by 66.9% (n = 121). However 17.1% (n = 31) selected the distractor "acute mitral insufficiency", 11.6% (n = 21) "a decrease in preload", and 3.3% (n = 6) "pericardial tamponade". Two subjects did not respond to this item. The question difficulty was 67 (medium), and the discrimination index was 0.23 (fair).

Item 11 required identification of appropriate nursing action in response to the problem identified in item 10. The correct response "have the patient turn on his side or move his arm" was selected by 42.5% (n = 77) subjects. However, 27.1% (n = 49) subjects selected the distractor "inflate the balloon to evaluate the PA wedge pressure", 24.3% (n = 44) chose "vigorously flush the system", and "wean the vasodilator therapy" was selected by 5.5%

(n = 10) of the subjects. Five individuals did not respond to this item this item. The item difficulty was 43 (hard), and the discrimination index was 0.18 (fair).

Item 12 required identification of PA systolic pressure at end-expiration in a mechanically ventilated patient. Clinical information to guide the decision was provided in a scenario. The correct response was selected by 57.5% (n = 104). The distractor that reflected PA systolic during a positive-pressure inspiration was selected by 33.7% (n = 61), while 6.6% (n = 12) selected the distractor that reflected PA wedge pressure. Two individuals did not respond to this item. The difficulty index was 57 (medium), and the discrimination index was 0.36 (good).

Item 13 required identification of the PA diastolic pressure at end-expiration. The correct response was selected by 57.5% (n = 104). The distractor that reflected PA diastolic during a positive-pressure inspiration was selected by 33.7% (n = 61), and 6.6% (n = 12) read the peak of the PA wedge pressure tracing. Two individuals did not respond to this item. The difficulty index was 61 (medium), and the discrimination index was 0.26 (fair).

Item 14 required identification of the PA mean pressure at end-expiration. The correct response was identified by 27.1% (n = 49). The distractor that reflected measurement at the dicrotic notch was selected by 34.8% (n = 63), 27.1% (n = 49) chose the dicrotic notch in the waveform that occurred during positive-pressure inspiration, and 9.4% (n = 17) chose the mean PA wedge pressure. Three

individuals did not respond to this item. The item difficulty index

was 27 (hard), and the item discrimination index was 0.37 (good).

Item 15 required identification of the PA wedge pressure at

end-expiration. The correct response was selected by 55.2%

(n = 100). The distractor that reflected the peak of the "a" wave on

the PA wedge pressure tracing was selected by 29.8% (n = 54), and

12.7% (n = 23) chose the distractor that reflected the lowest point

on the PA wedge pressure waveform. Distractor "A" had a

typographical error that did not reflect the waveform; therefore,

only three options were available. Three subjects did not respond to

this item. The difficulty index was 55 (medium), and the

discrimination index was 0.13 (fair).

Item 16 required analysis of the clinical data presented in the

scenario related to items 12 through 15, and the PA and PA wedge

pressures identified in items 12 through 15, in order to make a

diagnosis. The correct response "left ventricular failure" was

selected by 86.2% (n = 156). The distractor "mitral insufficiency"

was selected by 7.7% (n = 14), and "acute pericardial tamponade" by

3.9% (n = 7). Two subjects did not respond to this item. The

difficulty index was 86 (easy), and the discrimination index was 0.18

(fair).

Item 17 required synthesis of the data from items 12 through

16, and recognition of the clinical manifestations that were

consistent with these data. The correct response that reflected

recognition of both pulmonary congestion and impaired systemic

perfusion as consistent with the clinical data was selected by 91.7% (*n* = 166) of the subjects. Two subjects did not respond to this item. The item difficulty index was 92 (easy), and the discrimination index was 0.20 (fair).

Item 18 required the synthesis of information from items 12 through 17, and the correct diagnosis based on the information presented. Based on these data the subject developed a plan of care for the treatment of cardiogenic shock (systemic hypoperfusion and pulmonary congestion). The correct response "volume reduction" was selected by 58.0% (*n* = 105). The distractor "chronotropic therapy" was selected by 24.3% (*n* = 44), "vasoconstriction" was selected by 8.8% (*n* = 16), and 7.7% (*n* = 14) chose "volume augmentation". The item difficulty index was 58 (medium), and the item discrimination index was 0.38 (good).

Item 19 assumed the subject had correctly identified the clinical diagnosis and plan of care for the patient. The subject was asked to identify the goal for the therapy outline in item 18. The correct response "decrease in preload" was selected by 61.3% (*n* = 111). However 27.1% (*n* = 49) selected the goal for therapy as an increase in contractility, although there was no option in item 18 related to an increase in contractility. Four individuals did not respond to this item. The item difficulty index was 61 (medium), and the item discrimination index was 0.28 (fair).

Item 20 required identification of other therapeutic options for the the treatment of cardiogenic shock. The correct answer

(exception) " preload augmentation" was identified as an

inappropriate therapeutic option by 61.9% (n = 112). However, 22.7%

(n = 41) selected "vasodilator therapy", and 8.3% (n = 15) chose

"increase in contractility" as inappropriate therapy for cardiogenic

shock. Three subjects did not respond to this item. The item

difficulty index was 62 (medium), and the discrimination index was

0.31 (good).

Item 21 required knowledge of the parameters used to describe

left ventricular end-diastolic pressure. The correct response

(exception) "central venous pressure" was selected by 78.5%

(n = 142). The distractor "left atrial pressure" was selected by

16.0% (n = 29) of the subjects. Four subjects did not respond to

this item. The item difficulty index was 78 (medium), and the

discrimination index was 0.29 (fair).

Item 22 required knowledge of the parameters used to describe

cardiac work. The correct response (exception) "PA wedge pressure"

was selected by 54.1% (n = 98); however, 39.2% (n = 71) chose "stroke

work" as an incorrect index of cardiac index. Two subjects did not

resppond to this item. The difficulty index was 54 (medium), and the

discrimination index was 0.25 (fair).

Item 23 required knowledge of the effects of the therapy

outlined in Items 18 to 20, and the ability to utilize this

information to correctly interpret a ventricular function curve. The

correct response was selected by 82.9% (n = 150). The distractor

that indicated an increase in cardiac index secondary to improved

contractility, without reduction in preload was selected by 9.9%
(*n* = 18). Five individuals did not respond to this item. The item
difficulty index was 83 (medium), and the discrimination index was
0.34 (good).

Item 24 required analysis of a "PA pressure" waveform and
electrocardiogram tracing to identify a right ventricular pressure
waveform tracing. The correct response was selected by 83.4%
(*n* = 151). The distractor "pulmonary hypertension" was selected by
6.1% (*n* = 11), and "left ventricular fluid overload" was selected by
4.4% (*n* = 8). Five individuals did not respond to this item. The
item difficulty index was 83 (medium), and item discrimination was
0.33 (good).

Item 25 required correct identification of the waveform in item
24, and the development of an appropriate plan of care to resolve the
complication. The correct response "inflation of the PA catheter
balloon" was selected by 68.0% (*n* = 123). The distractor "assessment
of dynamic response" was selected by 18.2% (*n* = 33), and 7.2%
(*n* = 13) selected "administration of prn diuretic". Five individuals
did not respond to this item. The item difficulty index was 68
(medium), and item discrimination was 0.27 (fair).

Item 26 required that the subject to recognize the effect of
10 cm H_2O positive end-expiratory pressure (PEEP) had on PA wedge
pressure, and to correct the PA wedge pressure for the PEEP effect.
The correct response, which required subtraction of one-half the
applied PEEP (converted to torr) from the PA wedge pressure, was

selected by 33.7% (n = 61). The distractor that indicated no change

in PA wedge pressure was selected by 28.2% (n = 51), and the

distractor that reflected subtraction of the full amount of PEEP

(cm H_2O) was also chosen by 28.2% (n = 51). A further increase in PA

wedge pressure was selected by 7.2% (n = 13). Five individuals did

not respond to this item. The item difficulty index was 34 (hard),

and the item discrimination was 0.14 (fair).

Item 27 required knowledge of the effect a side-lying position,

with the backrest elevated, on the accuracy of PA pressure

measurements, and the ability to utilize this knowledge to interpret

a given set of clinical data. The correct response was selected by

65.2% (n = 118). However, 23.2% (n = 42) selected the distractor

"accurate because the transducer was referenced correctly to the

sternum". In addition, 8.3% (n = 15) chose the distractor

"reflective of a further decrease in cardiac function". Four

individuals did not respond to this item. The item difficulty index

was 65 (medium), and the discrimination index was 0.16 (fair).

Item 28 required identification of the rationale for the

interpretation of the data in item 27. The correct response "PA

pressure measurements are not consistently reproducible in the side-

lying position" was selected by 60.8% (n = 110). The distractor "PA

pressure measurements are consistently reproducible in all side-lying

positions, as long as the transducer air-fluid interface is correctly

referenced to the sternum" was selected by 29.3% (n = 53). Five

individuals did not respond to this item. The item difficulty index was 61 (medium), and the discrimination index was 0.18 (fair).

Item 29 required knowledge of the effect of position and position change on PA pressure measurement accuracy, and use of this information in developing a plan of care. The correct response "comparing flat, supine PA pressure measurements with supine, backrest upright pressure measurements for consistency" was selected by 45.3% (n = 82). However, 29.3% (n = 53) selected the distractor "placing him flat and supine for PA pressure measurements", and 19.3% (n = 35) chose "averaging PA pressure measurements over several respiratory cycles". Seven individuals did not respond omitted this item. The item difficulty index was 45 (hard), and the discrimination index was 0.16 (fair).

Analysis of Highest and Lowest Correct Response Items

The seven items (25%) that received the highest number of correct answers identified areas of widespread knowledge among the sample. Table 8 lists in rank order the seven items that received the most correct answers and the seven items that received the fewest correct answers. Items 4, 3, 17, 1, 16, 24, and 23 (in rank order from highest to lowest) were answered correctly most often. Item 4 confirmed that the subjects recognized the relationship between PA pressure and the occurrence of pulmonary congestion. Item 3 confirmed that the subjects were able to recognize pulmonary edema from the clinical and hemodynamic data that were provided. Item 17 confirmed that the subjects were able to identify the relationship

Table 8.

Seven Highest Scoring Items and Seven Lowest Scoring Items

Seven Highest Scores

Item Number	Options	Frequency	Percentage
4	A	4	2.2
	B	3	1.7
	C	0	0.0
	D*	173	95.6
	OMIT	1	0.6
3	A	0	0.0
	B	1	0.6
	C*	171	94.5
	D	9	5.0
17	A	0	0.0
	B	6	3.3
	C	2	1.1
	D*	166	91.7
16	A	7	3.9
	B	14	7.7
	C	2	1.1
	D*	156	86.2
1	A	9	5.0
	B*	155	85.6
	C	15	8.3
	D	2	1.1
24	A	11	6.1
	B	8	4.4
	C	6	3.3
	D*	151	83.4
23	A	3	1.7
	B	18	9.9
	C*	150	82.9
	D	5	2.8
	OMITS	5	2.8

Table 8 (continued)

Seven Lowest Scores

Item Number	Options	Frequency	Percentage
9	A	139	76.8
	B	1	0.6
	C*	39	21.5
	D	2	1.1
14	A	49	27.1
	B	63	34.8
	C*	49	27.1
	D	17	9.4
	OMITS	3	1.7
26	A	13	7.2
	B	51	28.2
	C*	61	33.7
	D	51	28.2
	OMITS	5	2.8
11	A	44	24.3
	B*	77	42.5
	C	10	5.5
	D	49	27.2
	OMITS	1	0.6
29	A	53	29.3
	B	4	2.2
	C*	82	45.3
	D	35	19.3
	OMITS	7	3.9
22	A	3	1.7
	B	71	39.2
	C*	98	54.1
	D	7	3.9
	OMITS	2	1.1
15	A	1	0.6
	B	54	29.8
	C*	100	55.2
	D	17	9.4
	OMITS	3	1.7

*Correct answer

between hemodynamic data and the presence or absence of pulmonary

congestion and systemic hypoperfusion. Item 1 confirmed that the

subjects could correctly define the term preload. Item 16 confirmed

that the subjects could make the diagnosis of left ventricular

failure based on hemodynamic and clinical data. Item 24 confirmed

that the subjects could correctly identify a right ventricular

waveform from a pressure tracing. Item 23 confirmed that the

subjects could correctly identify the desired effect of therapies for

cardiogenic shock, using a ventricular function curve. Items 3,4,16,

and 17 all represent content that is used daily in the management of

critically ill cardiovascular patients; therefore it was expected

that these items would be answered correctly by most of the subjects.

Items 9, 14, 26, 11, 19, 22, and 15 (in rank order from lowest

to highest correct response rate) received the seven lowest scores.

Item 9 tested knowledge of the term phlebostatic axis. More subjects

chose the distractor "... at the midaxillary line" than the correct

response. Item 14 tested the ability to identify mean PA pressure

from an analog tracing in a mechanically-ventilated patient. More

subjects chose the dicrotic notch in the end-expiratory waveform as

indicative of the mean pressure, than the actual mean pressure. In

addition, while 49% of the subjects answered the item correctly, 49%

incorrectly identified the PA mean pressure as occurring during

positive-pressure inspiration. Item 26 required the subjects to

determine the effect of PEEP therapy on the PA wedge pressure, and to

calculate the actual PA wedge pressure. A similar percentage of

subjects either subtracted the entire PEEP (cm H_2O) level, or made no change in the PA wedge pressure. Item 11 tested the subjects ability to recognize and determine a treatment plan for a spontaneously wedged catheter. While 66.9% of the subjects correctly identified the waveform as indicative of a wedged catheter (item 10), only 42.5% correctly identified the appropriate therapy. The distractor "inflate the balloon to evaluate the PA wedge pressure" was chosen by 27.1% of the sample, and 24.3% of the sample chose "vigorously flush the system". Item 29 tested knowledge related to the effect of position and position change on the accuracy of PA pressure measurements. The correct response "comparing flat supine PA pressure measurements with supine, backrest upright pressure measurements for consistency" was selected by 45.3%. However 29.3% chose the distractor "place him flat and supine for all pressure measurements", while 19.3% chose the distractor "averaging PA pressure measurements over several respiratory cycles". Item 22 tested the subjects knowledge of the clinical indices of cardiac index. Fifty five percent of the subjects correctly recognized that PA wedge pressure was not an index of cardiac work, however, 39.2% incorrectly identified "stroke work" as an incorrect index of cardiac work. Item 15 tested the ability to identify the PA wedge pressure on an end-expiratory analog tracing. While 55.2% chose the correct response, 29.8% identified the peak of the "a" wave as the pressure, and 12.7% chose the lowest pressure on the waveform as indicative of PA wedge pressure.

Differences in Total and Subset Scores Related to Selected
Demographic Characteristics

The differences in mean total and subset scores based on
selected demographic characteristics were analyzed using the
Statistical Package for the Social Sciences (SPSS) (Table 9). A one-
way analysis of variance (ANOVA) was used to determine if the test
scores for each of the demographic characterisitics were different.
If a significant difference ($p < 0.05$) between the means was
discovered, the Tukey B, a *post-hoc* multiple-comparison test, was
conducted to determine which demographic levels were different
relative to total and subset test scores. In order to avoid Type I
errors, if the ANOVA failed to detect a difference between the means,
any statistically significant differences that were identified by the
Tukey B were disregarded. A one-tailed, pooled *t*-test was used to
analyze the relationship between demographic variables with two
levels and the mean test scores. It was hypothesized that
individuals with additional certification (ACLS, CCRN) would score
higher than those who did not possess the certification. Data were
analyzed at a significance level of alpha = 0.05.

Age.

Age was not significantly ($p > 0.05$) related to any test score
except for Level 2 (application and analysis), where the 35 to 39
year age group (mean = 7.04) had a significantly higher score (ANOVA
$p = 0.0249$; Tukey B $p < 0.05$) than the 30 to 34 age group (mean =
5.40).

Table 9.

Summary of Statistical Analysis of Differences in Mean Test Scores

and Demographic Variables

Demographic Variables/Group	n	Total	Clin	Tech	Comp	Level 1	Level 2	Level 3
Total Scores		29.00	16.00	9.00	4.00	10.00	11.00	8.00
Age (N = 174)								
1 20-29	34	18.85	12.21	4.15	2.50	5.47	7.15	5.35
2 30-34	47	19.79	12.11	4.74	2.94	5.85	5.40	5.55
3 35-39	47	19.56	12.49	4.45	2.66	6.00	7.04	5.49
4 40-44	30	19.00	12.63	3.83	2.53	5.53	5.93	5.53
5 45+	16	16.56	10.50	4.06	2.00	4.50	5.75	4.44
ANOVA		.0799	.0826	.2252	.0867	.1315	.0249	.1602
Tukey B		NS	NS	NS	NS	NS	.05	NS
Comments							3 > 2	
State of Residence (N = 172)								
1 CT,ME,MA,NH,RI,VT	13	20.08	12.00	4.92	3.15	6.31	6.92	5.31
2 NY	9	18.33	12.00	3.89	2.44	5.67	7.44	5.22
3 DE,NJ,PA	24	17.58	11.04	4.38	2.17	4.83	6.63	4.88
4 DC,MD,VA,WV	11	20.36	13.09	4.36	2.91	5.82	4.18	5.82
5 NC,SC	5	19.00	11.80	4.20	3.00	4.40	6.00	4.60
6 AL,GA	8	18.38	12.25	4.00	2.13	5.75	8.00	4.63
7 FL,PR,GU	11	21.27	13.90	4.45	2.91	6.64	7.00	5.82
8 MI,WI	12	21.58	14.50	4.25	2.83	6.25	5.33	6.67
9 IN,OH	12	18.58	11.58	4.33	2.67	6.25	6.67	5.67
10 IL	6	18.50	11.67	4.83	2.00	4.83	8.00	5.67
11 KY,TN	6	21.00	12.33	5.33	3.33	6.17	5.33	6.17
12 AR,LA,MS	5	14.20	10.20	2.20	1.80	3.80	6.00	4.40
13 IA,MN,NE	3	20.67	14.67	3.00	3.00	5.67	8.33	6.67
14 KS,MO	7	19.86	11.43	5.14	3.29	6.57	6.57	5.29
15 OK,TX	13	18.61	12.77	3.23	2.62	5.46	6.38	5.23
16 MT,ND,SD,WY	2	22.00	12.50	6.50	3.00	6.00	4.00	7.00
17 AS,CO,NV,NM,UT	9	20.00	12.22	5.11	2.67	4.44	6.11	6.11
18 AK,ID,HI,OR,WA	5	18.60	12.40	4.20	2.00	4.40	5.40	4.80
19 CA	11	18.00	11.09	4.36	2.55	6.36	6.00	4.73
ANOVA		.1061	.0267	.2051	.3135	.1330	.4124	.0069
Tukey B		NS	.05	NS	NS	NS	NS	NS
Comments			8 > 3					

Table 9 (continued)

Demographic Variables/Group	n	Total	Clin	Tech	Comp	Level 1	Level 2	Level 3
Total Scores		29.00	16.00	9.00	4.00	10.00	11.00	8.00
Basic Education in Nursing (N = 173)								
1 Diploma	59	19.03	11.80	4.52	2.71	5.42	6.73	5.36
2 Associate	39	17.69	11.82	3.82	2.05	5.74	6.79	4.90
3 Baccalaureate or Higher	75	19.89	12.65	4.39	2.85	5.81	5.71	5.71
ANOVA		.0231	.1077	.1629	.0240	.5276	.0704	.0349
Tukey B		.05	NS	NS	.05	NS	NS	.05
		3 > 2			3 & 1 > 2			3 > 2
Highest Degree in Nursing (N = 172)								
1 Diploma	35	18.91	11.63	4.54	2.74	5.57	6.57	5.34
2 Associate	30	17.37	11.53	3.60	2.23	5.63	6.30	4.77
3 Baccalaureate	77	19.57	12.57	4.45	2.55	5.71	6.40	5.64
4 Master's	30	20.00	12.47	4.40	3.13	5.57	5.63	5.47
ANOVA		.0484	.1430	.1440	.0265	.9821	.6007	.0891
Tukey B		.05	NS	NS	.05	NS	NS	NS
Comments		4 > 2			3 > 2			
ACLS Provider (N = 177)								
1 Yes	123	19.21	12.33	4.31	2.57	5.65	6.08	5.44
2 No	52	18.90	11.81	4.33	2.77	5.60	6.88	5.27
One-tailed t-test		NS	NS	NS	NS	NS	.05	NS
Comments							1 < 2	
ACLS Instructor (N = 175)								
1 Yes	35	19.69	12.37	4.40	2.91	5.86	6.63	5.49
2 No	140	18.98	12.13	4.29	2.56	5.59	6.24	5.37
One-tailed t-test		NS	NS	NS	< 0.10	NS	NS	NS

Table 9 (continued)

Demographic Variables/Group	n	Total	Clin	Tech	Comp	Level 1	Level 2	Level 3
Total Scores		29.00	16.00	9.00	4.00	10.00	11.00	8.00
CCRN (N = 175)								
1 Yes	100	19.60	12.46	4.43	2.71	5.68	5.97	5.55
2 No	75	18.48	11.80	4.16	2.52	5.57	6.79	5.19
One-tailed t-test		.05	.05	NS	NS	NS	.05	.10
Comments		1 > 2	1 > 2			2 > 1	1 > 2	
Additional Certification (N = 175)								
1 Yes	30	18.30	11.20	4.37	2.73	5.57	6.03	5.03
2 No	145	19.29	12.38	4.30	2.61	5.65	6.38	5.47
One-tailed t-test		.10	.025	NS	NS	NS	NS	.10
		2 > 1	2 > 1					2 > 1
Years of Nursing Experience (N = 173)								
1 Less than 3	5	15.80	10.20	3.00	2.60	3.80	6.60	5.40
2 4 to 5	18	18.67	12.28	4.22	2.17	5.44	7.67	5.55
3 6 to 10	53	19.72	12.40	4.47	2.85	5.98	5.79	5.30
4 11 to 15	48	18.67	11.60	4.46	2.60	5.63	6.29	5.29
5 16 to 20	26	20.38	13.73	3.92	2.73	6.27	6.85	6.11
6 21 plus	23	18.30	11.57	4.39	2.35	4.83	5.83	5.04
ANOVA		.1263	.0058	.5025	.3193	.0320	.2333	.2246
Tukey B		NS	.05	NS	NS	NS	NS	NS
Comments			5 > 6 & 4					
Years of Critical Care Experience (N = 174)								
1 Less than 3	19	18.58	12.21	4.05	2.32	5.16	6.89	5.47
2 4 to 5	25	19.00	11.88	4.48	2.64	5.72	6.76	5.72
3 6 to 10	62	18.87	11.77	4.24	2.85	5.67	6.02	5.10
4 11 to 15	46	19.43	12.40	4.63	2.56	5.91	6.17	5.39
5 16 to 20	16	21.12	14.50	4.06	2.56	5.75	6.69	6.19
6 21 plus	6	18.50	12.00	4.17	2.33	4.83	6.67	5.33
ANOVA		.4065	.0085	.8024	.5407	.7219	.8020	.1999
Tukey B		NS	.05	NS	NS	NS	NS	NS
Comments			5 > 4, 3, 2 & 1					

Table 9 (continued)

<u>Demographic</u> Variables/Group	<u>n</u>	<u>Total</u>	<u>Clin</u>	<u>Tech</u>	<u>Comp</u>	<u>Level 1</u>	<u>Level 2</u>	<u>Level 3</u>
Total Scores		29.00	16.00	9.00	4.00	10.00	11.00	8.00
Years Since Active in Critical Care (N = 172)								
1 Less than 2	36	18.69	11.92	4.22	2.56	5.50	6.17	5.08
2 Greater than 2	16	17.00	11.50	3.38	2.13	5.00	6.75	4.63
3 Not applicable	120	19.50	12.34	4.44	2.72	5.77	6.33	5.57
ANOVA		.0568	.3866	.0950	.1682	.3368	.8059	.0399
Tukey B		NS	NS	NS	NS	NS	NS	NS
Employed in Nursing (N = 175)								
1 Full Time	129	19.32	12.23	4.39	2.70	5.61	6.19	5.42
2 Part Time	42	19.05	12.33	4.21	2.50	5.83	6.79	5.48
3 Other	4	13.50	8.75	3.00	1.75	4.25	5.50	3.75
ANOVA		.0185	.0276	.3153	.2185	.3347	.4569	.1127
Tukey B		.05	.05	NS	NS	NS	NS	NS
Comments		1 & 2 > 3	1 & 2 > 3					
Type of Facility (N = 175)								
1 Community	100	19.24	12.32	4.32	2.60	5.72	6.60	5.42
2 University	48	18.89	11.75	4.34	2.71	5.54	6.13	5.35
3 Other	27	19.07	12.41	4.07	2.59	5.48	5.63	5.37
ANOVA		.8906	.4093	.7204	.8652	.8138	.2787	.9698
Tukey B		NS	NS	NS	NS	NS	NS	NS
Hospital Size (N = 175)								
1 Less than 199	26	18.88	11.77	4.23	2.88	5.81	6.42	5.12
2 200 to 299	44	18.43	12.07	4.14	2.23	5.39	6.11	5/34
3 300 to 399	39	19.82	12.33	4.64	2.85	5.59	6.10	5.56
4 400 plus	57	19.75	12.51	4.49	2.75	6.00	6.47	5.53
5 Not applicable	9	16.11	11.11	2.89	2.11	4.22	7.00	4.89
ANOVA		.0661	.5165	.1119	.0443	.1444	.8995	.6380
Tukey B		NS	NS	NS	NS	NS	NS	NS

Table 9 (continued)

Demographic Variables/Group	n	Total	Clin	Tech	Comp	Level 1	Level 2	Level 3
Total Scores		29.00	16.00	9.00	4.00	10.00	11.00	8.00
Area of Employment (N = 175)								
1 Combined ICU/CCU	48	18.75	11.90	4.25	2.60	5.67	6.25	5.17
2 ICU	17	19.00	11.71	4.41	2.88	4.82	5.94	5.29
3 CCU	19	19.68	12.69	4.10	2.89	5.32	6.47	5.79
4 Surgical ICU	13	18.46	12.15	4.08	2.23	6.08	6.69	4.92
5 Cardiovascular-Surgical ICU	19	19.79	13.00	4.32	6.00	2.47	5.95	5.79
6 Rotate ICU	7	21.29	13.57	4.57	3.14	6.00	6.43	6.00
7 Other	52	18.92	11.92	4.44	2.56	5.71	6.10	5.38
ANOVA		.7226	.4027	.9886	.5597	.6280	.9343	.4882
Tukey B		NS	NS	NS	NS	NS	NS	NS
Position (N = 175)								
1 Staff/General Duty	91	19.12	12.03	4.38	2.70	5.78	6.40	5.41
2 Charge/Team Leader	20	18.80	12.45	4.00	2.35	6.00	7.20	5.10
3 Head/Asst Head Nurse	16	18.18	11.94	3.56	2.69	4.81	6.13	5.38
4 Inservice/Staff Development	11	20.27	12.45	5.27	2.54	6.55	5.45	5.55
5 CNS/Nurse Specialist	16	21.81	13.88	4.88	3.06	5.50	4.88	6.25
6 Academic Instructor	6	18.50	13.17	3.17	2.17	5.06	8.00	5.00
7 Other	15	17.07	10.53	4.27	2.27	5.13	6.40	4.87
ANOVA		.0448	.0232	.1226	.4432	.2467	.1980	.2910
Tukey B		.05	.05	NS	NS	NS	NS	NS
Comments		5 > 1	5 > 1					
Knowledge (N = 173)								
1 Novice	9	13.67	9.22	2.22	2.22	4.00	6.44	3.22
2 Advanced Beginner	14	17.50	11.21	4.07	2.21	5.29	6.00	4.79
3 Competent	79	19.16	12.15	4.37	2.65	5.71	6.52	5.42
4 Proficient	60	19.68	12.67	4.37	2.65	5.73	6.37	5.58
5 Expert	11	22.00	13.18	5.73	3.09	6.55	5.27	6.55
ANOVA		.0000	.0014	.0010	.3650	.0779	.7486	.0000
Tukey B		.05	.05	.05	NS	.05	NS	.05
Comments		5,4 &3 > 1; 5 > 2	5,4 & 3 > 1	5,4,3, & 2 > 1		5 > 1		5,4,3, & 2 > 1; 5 > 2

Table 9 (continued)

Note. Clin = clinical; tech = technical; comp = complications; Level 1 = knowledge and

comprehension; Level 2 = applications and analysis; Level 3 = synthesis and evaluation. ANOVA =

analysis of variance; ACLS = advanced cardiac life support; CCRN = critical care registered nurse.

Comments: refers to group differences ($p < 0.05$), i.e. group 1 > group 2

Region.

Individuals from region 8 (Michigan, Wisconsin) scored significantly higher (mean = 14.50/16.00, ANOVA p = 0.267, Tukey B p < 0.05) than individuals from region 3 (Delaware, New Jersey, Pennsylvania, mean = 11.04/16.00) on the Clinical subset. There was a trend toward significance (ANOVA p = 0.0069) on the Level 3 (synthesis and evaluation) subset, but no significant (p > 0.05) pair differences were noted. However, small sample sizes in each level limited statistical analysis of these data.

Basic education in nursing.

Nurses with basic education at the baccalaureate level had significantly higher scores on the total test (mean 19.89/29.00, ANOVA p = 0.0231, Tukey B p < .05) and Level 3 subset (mean = 5.71/8.00, ANOVA p = 0.0349, Tukey B p < 0.05) than individuals with basic education at the associate degree level (mean = 17.69/29 and 4.90/8, respectively). In addition, both baccalaureate (mean = 2.85/4) and diploma (2.71/4) nurses scored higher on the Complications subset (ANOVA $\dot{p}$ = 0.0240, Tukey B p < 0.05) than associate degree nurses (mean = 2.05/4). There were no differences between diploma and baccalaureate nurses on any test.

Highest education in nursing.

Nurses with a master's degree received the highest total test score (mean = 20.00/29). There was a significant difference between baccalaureate (mean = 19.57/29, ANOVA p = 0.0484; Tukey B p < 0.05) and associate degree nurses (mean = 17.37/29) on the total test

score. There was also a significant difference on the Complications subset score between nurses with master's (mean = 3.13/4; ANOVA p = 0.0265, Tukey B $p < 0.05$) and associate degree nurses (mean 2.23/4). There was a trend toward significance (ANOVA p = .0891) on Level 3 where baccalaureate nurses had a mean score of 5.64/8 and associate degree nurses had a mean score of 4.77/8. Associate degree nurses received the lowest test scores on five of seven tests, while nurses with baccalaureate degrees had the highest test scores on four of seven tests.

Certification.

Individuals who were ACLS providers scored lower (mean 6.08/10, t-test p < 0.05) than individuals who were not providers (mean 6.88/10) on the Level 2 subset; however, there were no significant differences on any other tests. Individuals who were ACLS instructors did not score signficantly higher ($p > 0.05$) on any test compared with individuals who were not ACLS instructors, or with ACLS providers. Individuals who had additional certifications (BLS, CEN) scored lower on the Clinical subset (mean = 11.20/16; $p < 0.025$) than individuals without additional certification (12.38/16). In addition, there was a trend toward significantly higher ($p < 0.10$) scores on the total test and the Level 3 subset for individuals without additional certifications.

Critical care registered nurse (CCRN).

Individuals who were CCRN certified had significantly higher ($p < 0.05$) total test (mean 19.6/29 versus 18.48/29), and clinical

subset (12.46/16 versus 11.80/26) scores. There was also a trend toward significance ($p < 0.10$) on the Level 3 subset. However, individuals who did not have CCRN certification scored significantly higher ($p < 0.05$) on the Level 2 subset (mean 5.97/11 versus 6.79/11). The CCRNs also scored higher on the Technical and Level 1 subsets, but the differences were not significant.

Years of nursing experience.

Nurses with 16 to 20 years of experience received the highest total test score (mean 20.38/29) and individuals with less than three years the lowest total test score (15.80/29); however, the differences were not significant ($p > 0.05$). Individuals with 21-plus years of nursing experience had the second lowest test score on five of seven tests, and the lowest score on the Level 3 subset, while individuals with less than three years had the lowest scores on four of seven tests. There was a significant difference (ANOVA $p = 0.0058$; Tukey B $p < 0.05$) between the 16 to 20 year group score on the Clinical subset (mean = 14.50/16) and the 21 plus (mean = 11.57/16) and 11 to 15 (mean 11.60/16) year groups. In addition, the group with less than three years of nursing experience ($n = 5$) had a mean score of 10.20/16, but the difference was not significant. There was a significant difference (ANOVA $p = 0.0320$) among the means on the Level 1 subset, however, there were no significant pair differences (Tukey B $p > 0.05$).

<u>Years of critical care experience</u>.

Nurses with 16 to 20 years of critical care experience received the highest total test score (21.12/29), but the difference with the other year groups was not significant (ANOVA p = 0.4065). In addition, the 16 to 20 year group had a significantly higher score 14.50/16) (ANOVA p = 0.0085; Tukey B p < 0.05) (mean 14.50/16) than any other group on the Clinical subset. There were no other significant differences.

<u>Years since active in critical care</u>.

There was a significant difference among groups on the total test (ANOVA p = 0.0568) and Level 3 subset (ANOVA p = 0.0399), but no significant (Tukey B p > 0.05) pair differences. Individuals who were active in critical care scored higher on all tests compared with individuals who were not currently active. Statistical analysis of these data was limited due to the small number of subjects in the group classified as not currently active.

<u>Employment status</u>.

There was no significant difference between individuals who were employed full-time versus part time in nursing. Both full time and part-time individuals scored significantly higher than individuals not currently employed in nursing on the total test (ANOVA p = 0.0185; Tukey B p < 0.05) and Clinical subset (ANOVA p = 0.276; Tukey B p < 0 05).

<u>Type of facility, facility size, and area of employment</u>.

There was no significant difference in test scores based on type of facility. There was a significant difference among groups based facility size on the Complications subset (ANOVA $p = 0.0443$), and a trend toward significance on the total test score (ANOVA $p = 0.0661$), but there were no significant (Tukey B $p > 0.05$) pair differences. There was no significant difference on test scores based on area of employment. However, individuals who rotated among the ICUs (including CNSs and staff development coordinators) received the highest scores on the total test (21.29/29), Clinical, Technical, and Level 3 subset.

<u>Position</u>.

Clinical nurse specialist's (CNSs) had the highest total test (21.81/29) and Clinical subset (13.88/16) scores. The CNS scores were signficantly higher (ANOVA $p = 0.0448$; Tukey B $p < 0.05$) than the scores for the general duty nurses. The CNSs also scored highest on the Complications (3.06/4), and Level 3 (6.25/8) subset. The inservice/staff development nurses received the highest scores on the Technical (5.27/9) and Level 1 (6.55/10) subset.

<u>Perceived knowledge of pulmonary artery pressure measurement</u>

There was a clear rank-wise increase in test scores based on perceived knowledge level. The individuals who rated themselves as novice received the lowest scores on all of the tests, while those individuals who rated themselves as expert received the highest scores on all of the tests. The experts received a mean total test

score of 22 of 29, while the novices' mean score was 13.67. Only the mean score for experts exceeded 70% correct; however, there was a wide range in the raw scores among the experts.

Summary of Findings

One thousand questionnaires were randomly sent to critical care nurses who were members in AACN, with 181 (18.1%) returned for data analysis. The data included a total test score, six subset scores, and demographic information. The demographic data from the sample were compared with the AACN population parameters, using the Chi-square test of proportions, to determine if the sample was representative of the target population. The test score data were analyzed for measures of central tendency (mean, median, mode) and dispersion (range and standard deviation). Each test item was analyzed using ScorePak for item option selection, item difficulty, and item discrimination. The demographic data with more than two levels were correlated with each of the seven test scores using a one-way analysis of variance and the Tukey B *post hoc* multiple comparison test, while the demographic variables with two levels were analyzed using a one-tailed pooled t-test.

The comparison of the sample demographic data with the AACN population parameters showed similarities between the sample and the population with regard to region, basic nursing education, hospital type, and area of employment. There were more nurses in the sample with the following characteristics: age in the 35 to 39 and 40 to 44 year groups, masters degree in nursing, employed full or part-time in

nursing, employed for greater than 11 years in critical care, and
employed as staff development or inservice coordinator or as a CNS.
There were fewer nurses in the sample with less than three years
experience in both nursing and critical care, as well as employment
in hospitals with greater than 400 beds. The nurses in the sample
perceived their knowledge level of PA pressure measurement as
competent (44.6%) or proficient (33.9%). The majority of the sample
were ACLS providers (70.6%), and had completed CCRN certification
(56.5%).

The scores of the total test had a relatively normal
distribution. The mean (18.98), median (19.00), and mode (20.00)
were similar. The negative skewness (-0.257) of the total test score
indicated there were more scores above the mean, as demonstrated by
the measures of central tendency. The leptokurtotic shape of curve
(kurtosis = 0.253) indicated there was slightly less variance in the
scores relative to the variance expected with a normal curve. In a
normal curve, 68% of the sample falls within one standard deviation
of the mean, 95% fall within 1.96 standard deviations of the mean,
and 99% fall within 2.56 standard deviations of the mean. In this
study, 72.92% of the sample fell within one standard deviation of the
mean, 96% fell within 1.96 standard deviations, and 99.5% fell within
2.56 standard deviations of the mean. This dispersion of test scores
is consistent with the leptokurtotic curve. The curve distribution
for this study was consistent with a norm-referenced test; however,

in a criterion-referenced test a negatively skewed curve was expected.

The mean scores for the subsets ranged from 48% to 71%, with a wide range of individual scores. There was a significant ($p < 0.05$) relationship between four of six subset scores (clinical, cognitive level 1, cognitive level 2, and cognitive level 3) and the total test score. Correlation between the technical subset score and the total score was high ($r = 0.71$), but did not reach statistical significance.

Item analysis was completed using ScorePak from the Educational Testing Service at the University of Washington. Item difficulty is the percentage of subjects who answered the item correctly, with a range of 1 to 100. For a multiple choice item, the ideal difficulty was 74. Five items were classified as easy, 19 items were classified as medium, and five items were classified as hard.

Item discrimination refers to the ability of an item to differentiate among subjects on the basis of how well they know the material being tested. One item was classified as a "poor", 22 items were classified as "fair", and six items were classified as "good" discriminators.

Comparison of the demographic data with the total and subset scores demonstrated wide variance in scores based on demographic characteristics. There were no significant ($p > 0.05$) differences in test scores based on age, region, certification status (ACLS or additional certification), years since active in critical care

practice, type of facility where employed, hospital size, or area of employment.

Nurses with basic education at the baccalaureate level consistently scored higher than associate degree nurses; there was no statistically signicant difference between the scores of diploma and baccalaureate nurses. Nurses with their highest education at the masters level scored significantly (ANOVA $p = 0.0484$; Tukey B $p < 0.05$) higher on the total test than associate degree nurses, while baccalaureate nurses scored significantly (ANOVA $p = 0.0265$; Tukey B $p < 0.05$) higher than associate degree nurses on the Complication subset. There was no significant ($p > 0.05$) difference between masters, baccalaureate, and diploma nurses on any of the tests. Individuals with CCRN certification scored significantly ($p < 0.05$) higher on four of seven tests.

Individuals with 16 to 20 years of nursing experience scored higher on four of seven tests, and individuals with less than three years of nursing experience scored lowest on four of seven tests; however, there was no consistent difference on test scores based on years of nursing. Similar results were found with regard to total years of critical care experience. Nurses with 16 to 20 years of experience scored highest on three of the seven tests, and were significantly higher (ANOVA $p = 0.0058$; Tukey B $p < 0.05$) than all other groups on the Clinical subset. However, nurses with 21 plus years of experience received the lowest scores on three of seven scores (including the total test).

There was no significant difference ($p < 0.05$) between nurses who were employed full-time versus part- time; however both groups scored higher than those nurses not currently employed in nursing on all tests. The CNSs scored significantly higher (ANOVA $p = 0.0448$, Tukey B $p < 0.05$) than staff nurses on the total test (21.82 versus 19.12) and Clinical subset (13.88 versus 12.03; ANOVA $p = 0.0232$; Tukey B $p < 0.05$), and had the highest score on four of seven tests. Once again, there was no cons stent difference based on practice position and test scores. The only consistently demonstrated differences on test scores with respect to demographic categories was the variable related to the preception of knowledge related to PA pressure measurement. Those individuals who rated themselves as novice scored the lowest on all of the tests, while those individuals who rated themselves as expert scored the highest. It is important to note, however, that there were wide ranges of scores in the competent, proficient, and expert groups.

A cautious profile of the individual who would score higher on on this test of knowledge and ability to utilize information related to PA pressure measurement, was the critical care nurse with 16 to 20 years of nursing and critical care experience, a baccalaureate or masters degree, CCRN certification, employment as a CNS or staff development coordinator, and a self-rating of expert with regard to PA pressure measurement. Caution must be taken when using this profile, because there were wide variances within each demographic category with regard to total and subset scores.

CHAPTER V

Discussion

This chapter presents a summary of the study's findings and
presents a discussion of the implications for nursing practice.
Limitations of the study and recommendations for further study are
also addressed.

Summary of the Study

The purpose of this study was to describe critical care nurses'
knowledge and ability to utilize information related to pulmonary
artery (PA) pressure measurement and to describe the extent that
relevant demographic data (age, experience in nursing, experience in
critical care, hospital size, area of employment, basic and advanced
education level, additional certification, and perceived knowledge
related to PA pressure measurement) correlated with the mastery of
this information.

The data-generating instruments were a 29-item, self-
administered, criterion-referenced questionnaire and a demographic
data sheet. The criterion items for the questionnaire were based on
a review of the literature related to PA pressure measurement. The
questionnaire was mailed to 1,000 randomly selected members of the
American Association of Critical Care Nurses (AACN). The
questionnaire packet included a cover letter explaining the purpose
of the study, demographic data sheet, the questionnaire (Clinical
Simulation: Pulmonary Artery Pressure Measurement), a mark sense

answer sheet, and a stamped return envelope. To increase the return

rate a follow-up postcard was sent ten days after the mailing of the

questionnaire packet, encouraging individuals to complete the data

gathering instruments. To ensure anonymity, the subjects were

instructed not to place their name on any of the forms, except for a

request for the test answers and abstract. The latter form was

separated from the demographic data sheet and mark sense answer sheet

prior to scoring. In addition, to ensure that the test reflected

only current knowledge, the subjects were asked not to use any

references to complete the tool.

A total of 181 (18.1%) mark sense answer sheets were returned

within six weeks of the initial mailing. Four individuals did not

return the demographic data sheet. One hundred and fifty subjects

requested abstracts and the rationale for the correct answers. Data

from the 181 mark sense answer sheets were analyzed using ScorePak

from the Office of Educational Assessment at the University of

Washington. The demographic data were computer analyzed and

correlated with the test scores using the Statistical Package for

Social Sciences (SPSS).

Comparison of the sample with the target population was

completed (Table 5). The characteristics of the sample revealed the

following: female (93.2%), age 30 to 34 (26.7%), basic education in

nursing at the baccalaureate level (43.80%), and highest degree in

nursing at the baccalaureate level (45.4%). A majority of the sample

were advanced cardiac life support (ACLS) providers (70.6%), and had

completed critical care registered nurse (CCRN) certification
(56.5%). Most subjects had six to ten years of experience in nursing
(30.30%) and in critical care (35.20%). The majority of sample were
active in critical care (70.2%), and employed full time in nursing
(74.00%). In addition, a majority worked in community hospitals
(57.10%) with 400 plus beds (32.80%). Most of the subjects worked in
combined intensive care/coronary care units (27.70%) as staff or
general duty nurses (52.50%). Most of the subjects rated their
knowledge of PA pressure measurement as competent (44.60%) or
proficient (33.90%). The subjects were distributed in a manner
consistent with the AACN population with respect to geographic
region.

When the sample was compared to the AACN population, there were
more nurses in the 35 to 39 and 40 to 44 year age groups, with
masters degrees in nursing, employed full or part-time in nursing,
employed for greater than 11 years in critical care, and employed
either as a staff development or inservice coordinator or as a
Clinical Nurse Specialist (CNS). There were fewer nurses in the
sample with less than three years of experience in both nursing and
critical care, as well as employment in hospitals with greater than
400 beds.

Each test item was classified into two categories: 1) content
area (clinical, technical, or complications), and 2) cognitive level
(level 1 - knowledge and comprehension, level 2 - application and
analysis, or level 3 - synthesis and evaluation). The item scores

for the total test and the six subset scores were analyzed for measures of central tendency (mean, median, and mode), and dispersion (range and standard deviation). The total test had a relatively normal distribution with a mean of 18.98 (65%), median of 19.00, and a mode of 20.00. The range of scores for the total test was 5 to 28 (17% to 96.6%). The standard deviation was 4.11 (14%), and 96% of the sample fell within 1.96 standard deviations of the mean. There were 16 items in the Clinical subset, with a mean score of 12.09 (75.6%), and a range of 5 to 16 (31% to 100%). There were nine items in the Technical subset, with a mean score of 4.28 (47.6%), and a range from zero to eight (0% to 89%). There were four items in the Complications subset, with a mean score of 2.61 (65%), and a range of zero to four (0% to 100%). The cognitive level 1 subset (knowledge and comprehension) consisted of ten items, with a mean score of 5.82 (58%), and a range from one to ten (0% to 100%). The cognitive level 2 subset (application and analysis) consisted of 11 items, with a mean score of 7.79 (71%), and a range of 2 to 11 (18% to 100%). The cognitive level 3 subset (synthesis and evaluation) consisted of eight items, with a mean score of 5.36 (67%), and a range from one to eight (12.5% to 100%).

Individual test items were analyzed as to how frequently they were chosen by the subjects, item difficulty, and item discrimination (Table G-4). In addition, the items in the top and bottom 25% with respect to frequency of correct responses were identified (Table 8).

Significant differences in the total and subset scores with demographic data sets were determined using one-way analysis of variance, Tukey B *post hoc* multiple comparison test, and one-tailed, pooled *t*-test. Statistical significance was identified as alpha = 0.05. There was a wide variance in the differences between demographic characteristics and total and subset scores. The only consistent difference noted was between an individual's perceived knowledge level and their test scores. Individuals who rated themselves as novice had the lowest scores on all seven tests, while individuals who rated themselves as expert had the highest scores on all of the tests. However, the "experts" had a wide range of scores, e.g., their mean total score was 22 (76%), with a range from 16 to 28 (55% to 97%), while the "novices" had a mean score of 13.67 (47%), with a range from 5 to 19 (17% to 66%). Nurses with a baccalaureate or masters degree consistently scored higher than associate degree nurses, but the differences between diploma, baccalaureate, andor masters prepared nurses were not significant. Individuals with CCRN certification scored significantly higher ($p < 0.05$) on four of seven tests. Nurses with 16 to 20 years of nursing experience scored higher on four of seven tests, while individuals with less than three years of experience scored lowest on four of seven tests; however, there were no consistent differences in test scores based on years of nursing experience. Similar results were noted with regard to years of critical care experience. Nurses with 16 to 20 years of experience scored highest on three of seven

tests; however, nurses with 21 plus years of experience scored lowest

on three of seven tests (including the total test). Clinical Nurse

Specialists had the highest scores on four of seven tests, and staff

development coordinators on two of seven tests, but there were no

differences in test scores based on position. The CNSs mean total

test score was 21.81 (75%), with a range from 14 to 28 (48% to 97%),

and the staff development coordinators' mean total score was 20.27

(70%), with a range from 13 to 28 (45% to 97%). Staff nurses, the

majority of the sample, had a total mean of 19.12 (66%), with a range

from 8 to 27 (27.6% to 93%). Therefore, while certain demographic

characteristic were associated with higher or lower test scores, wide

individual variations within each characteristic level occurred.

Implications for Nursing

Pulmonary artery pressure measurement is used in 90% of all

adult critical care units (Holmes, 1982). The safe, effective use of

the PA catheter and the hemodynamic data provided by the system

requires an extensive knowledge base. Lack of knowledge related to

recognition of pathognomonic alterations in hemodynamic parameters,

complications associated with PA pressure measurement, or use of

incorrect technique may result in therapeutic mismanagement or injury

to the patient. Pulmonary artery pressure measurement is commonly

used to monitor patients with complicated myocardial infarctions. In

these patients, an alteration in compliance, pulmonary vascular

resistance, synergy of contraction, or heart rate may affect the

relationship between PA pressures and left ventricular pressures.

Because of this alteration in the PA-left ventricular pressure relationship, every effort must be made to recognize the effects of the pathophysiologic changes on the hemodynamic values, and to attend to the numerous technical variables that may decrease the accuracy of PA pressure measurement as an indicator of left ventricular function.

The mean total test score on the Clinical Simulation: Pulmonary Artery Pressure Measurement was 18.98 (65.4%), with a range of 5 to 28. Only 34% of the sample scored above 70% (20.3 items). This mean score is interpreted as indicative of a general knowledge deficit related to PA pressure measurement. Analysis of the subset scores provided a more specific identification of the knowledge deficit. The study data suggested a general knowledge deficit in each content area; however, the Technical subset score of 4.28 (47.5%) was the lowest. There were also knowledge deficits in all of the cognitive levels; however, Level 1 (knowledge and comprehension) had the lowest score 5.82 (58.2%). Further evaluation of the individual test items confirmed both the general and specific knowledge deficits.

Data from two nation-wide studies of critical care nurses' knowledge related to PA pressure measurement were compared with the data from this study to identify consistent knowledge deficits. In this study, the items that received the fewest correct responses indicated a knowledge deficit of the most basic information related to PA pressure measurement.

Item 9 tested knowledge of the term phlebostatic axis. A majority of the subjects (76.8%) chose the distractor "intersection

of an axis transsecting the fourth intercostal space at the sternum and the midaxillary line", as opposed to the correct response of one-half the anterior-posterior chest diameter. In a study by Bartz and coworkers (1988), the use of the midaxillary line as opposed to the mid-anterior-posterior level resulted in a pressure difference of up to 6-torr. In a critically ill patient, a 6 torr pressure difference, introduced by use of an incorrect reference level, may result in therapeutic mismanagement. Straw's study (1986) also identified incorrect referencing as a knowledge deficit, while Dolter (1987) identified the phlebostatic axis as the midaxillary line, and did not offer one-half the anterior-posterior diameter as an option.

Items 14 and 15 required the subject to identify the end-expiratory mean PA and PA wedge pressures from an analog tracing. More subjects (34.8%) chose the dicrotic notch in the end-expiratory waveform as indicative of the mean pressure than the actual mean pressure (27.1%). The PA wedge pressure was correctly identified by 55.2% of the subjects; however, 29.8% identified the peak of the "a" wave as the correct pressure. In addition, the PA systolic pressure (item 12) was only identified correctly by 57.5%, while 33.7% read the PA systolic pressure during the positive-pressure inspiration. Similar results were found relative to the PA diastolic pressure (item 13). Current technology allows for identification a desired waveform with a cursor on the oscilloscope, with the pressure provided in a digital form. However, if the selection of waveforms is incorrect, the measured pressure will be in error. In addition,

evaluation of the accuracy of digital data requires the ability to

first read the analog waveform to ensure that the correct pressures

are being "read" by the monitor. Finally, the monitor does not

possess the capability to "know" whether a patient is breathing

spontaneously or is being mechanically-ventilated. Therefore, the

nurse must be able to interpret digital data relative to this

clinical data. Both Straw (1986) and Dolter (1987) found that nurses

knew that PA pressures should be measured at end-expiration, but that

they were unable to consistently apply this information when given

analog waveforms from either spontaneously breathing or mechanically-

ventilated patients.

Item 26 required correction of a PA wedge pressure following

application of 10 cm H_2O positive end-expiratory pressure (PEEP).

Only 33.7% of the subjects correctly subtracted one-half the applied

PEEP, converted from cm H_2O to torr from the PA wedge pressure. Left

ventricular distention, which is caused by transmural pressure, is

the the value of interest when evaluating left ventricular function.

Intrapleural pressure is assumed to be equal to atmospheric pressure;

however, the application of PEEP increases the intrapleural pressure.

Therefore, in the presence of PEEP, accurate measurement of the PA

wedge pressure (the indicator or left ventricular distention)

requires subtraction of an estimate of pleural pressure. While

trends in pressure changes are generally used to guide therapy,

individual pressure measurements may also be used, e.g, a PA wedge

pressure of 22 torr. If the effect of PEEP is not recognized and

corrected for, once again therapeutic mismanagement may occur.
Dolter (1987) also found that critical care nurses did not recognize
the effect of PEEP on the accuracy of PA wedge pressure as an index
of left ventricular end-diastolic pressure.

Item 11 tested the subjects' ability to recognize and treat a
spontaneously-wedged catheter. While 66.9% of the subjects correctly
identified the waveform as indicative of a spontaneous wedge (item
10), only 42.5% were able to correctly identify the appropriate
treatment. The distractor "inflate the balloon to evaluate the PA
wedge pressure" was selected by 27.1% of the sample, and 24.3% chose
to "vigorously flush the system". Spontaneous wedging of the
catheter indicates catheter migration into a distal pulmonary vessel.
Failure to resolve a spontaneous wedge may result in pulmonary
infarction secondary to vessel occlusion. The correct response was
to reposition the catheter. In addition, gentle flushing of the
system may be useful to differentiate a damped waveform, due to a
microthrombi on the catheter tip, from a true wedge. However,
inflation of the balloon or vigorous flushing of the catheter in a
small pulmonary vessel may result in the catastrophic rupture of the
pulmonary vessel. Dolter's (1987) study found that 35% of the sample
subjects could not correctly identify the treatment for a spontaneous
wedge.

Item 29 tested knowledge related to the effect of position and
position change on the accuracy of PA pressure measurements. The
correct response "compare flat, supine PA pressure measurements with

supine, backrest upright pressure measurements for consistency" was selected by 45.3% of the sample, while 29.9% chose the distractor "place him flat for all pressure measurements". While the distractor is not incorrect, use of the flat position may be poorly tolerated by patients with pulmonary or cardiovascular compromise. There is sufficient research to indicate that PA and PA wedge pressures can be attained in the supine position with a backrest elevation up to 60-degrees, as long as the upright pressures are evaluated and found to be consistent with flat, supine pressures. In addition, placement of the patient in the flat, supine position does not provide accurate information related to the patient's hemodynamic status in any other position. Straw's (1986) study identified 20 degrees as the maximum backrest elevation.

Item 22 tested the subjects' knowledge of the clinical indices of cardiac index. While 55% of the sample recognized that PA wedge pressure was not an index of cardiac work, 39.2% identified stroke work as the incorrect index. These responses appear to reflect a lack of knowledge related to terminology. Correct use of a ventricular function curve requires knowledge of the parameters being utilized, as well as how they are altered as a result of pathology or therapy.

Straws's study found there was a knowledge deficit related to measuring PA pressures in the 90- degree lateral-recumbent position. Only 12% of Straw's sample were able to identify the reference level for the 90-degree lateral position, while 46% indicated that PA

pressure measurement should never be undertaken in the lateral recumbent position. Item 27 in this study tested knowledge related to the effect of a 30-degree side-lying position, with the backrest elevated, on the accuracy of PA pressure measurements. Item 28 asked for the rationale for the answer in item 27. While 65% and 61% of the subjects chose the correct answer and rationale, respectively, 23% and 29%, indicated that PA pressure measurement in the side-lying position was accurate as long as the transducer was correctly referenced to the sternum. In addition, 8.3% indicated that the pressure changes were consistent with a decrease in cardiac function. Nine studies were found that evaluated the effect of lateral positioning on PA pressure measurement accuracy. Based on findings from these studies, the only position that has been shown to provide reliable PA and PA wedge pressures relative to the flat, supine position is 90-degrees lateral with 0-degree backrest elevation. These two items demonstrate how incorrect technique may result in inappropriate treatment, as well as the lack of dissemination of research related to the effect of positioning on PA pressure measurement accuracy.

Four of the seven items that were answered correctly most often were related to the recognition of relationships between hemodynamic data and clinical presentation. The other three items were related to recognition of a right ventricular tracing, the definition of preload, and the ability to identify the desired effect of specified therapies on a ventricular function curve.

The items answered incorrectly most often were reflective of the most basic knowledge related to PA pressure measurement, e.g., reading an analog pressure tracing, knowledge of ventilatory effects, recognition of the effect of position and position change on pressure measurement accuracy, the correct referencing of the PA catheter system, and how to resolve a potentially dangerous complication associated with PA pressure measurement. These test results demonstrate a disturbing fact; while critical care nurses are able to use hemodynamic data once it has been collected, they often utilize incorrect technique in gathering the data. The use of incorrect technique introduces error, and may in fact invalidate the hemodynamic data. The end-result of erroneous data collection may be the use of potentially invalid data to guide therapy. Three nation-wide studies have been conducted over a six-year period, and all three have demonstrated a similar knowledge deficit related to the technical aspects of PA pressure measurement.

The demographic characteristics of the study indicated that the general knowledge deficit related to PA pressure measurement was identified with critical care nurses at all levels of practice. While the staff development coordinators and CNSs received the highest test scores, neither group exceeded 75%. Within each of these two groups there was a wide range of scores, indicating that the individuals who are often consulted for problems related to PA pressure measurement do not possess an accurate knowledge base. Of equal importance, the staff nurses who are most often responsible for

the collection, interpretation, and utilization of PA pressure

measurement data had a total mean score of 66%, 75% on the Clinical

subset, and 49% on the Technical subset. These results are of

concern because both the nurses who perform PA pressure measurement

on a day-to-day basis, and those who are consulted to assist them, do

not demononstrate a consistent knowledge base or the ability to

utilize information related to PA pressure measurement.

It was expected that individuals with a higher educational

level, more years of experience, or CCRN certification, which is

indicative of possession of a knowledge base related to the practice

of critical care nursing, would have higher test scores. While

nurses with these demographic characteristics did score higher on

many of the tests, there were no consistent differences between test

scores based on specific demographic variables. These findings are

consistent with the congruence in inference and action studies

conducted by Keihm (1985), Edgers (1987), Winston-Heath (1988), and

Sims (1989), and the PA pressure measurement study conducted by Straw

(1986). These studies found a difference in the frequency of

congurence between inference and action in the care of critically ill

patients based on nursing education and nursing experience; however,

the differences were inconsistent. Straw (1986) found a significant

difference ($p < 0.05$) in test score based on CCRN certification and

age (45 to 49), but not educational level or years of experience.

There are several possible explanations for these findings.

While nurses who have been practicing longer should have more

experience in using PA pressure measurement data, these nurses may be in management positions where they no longer utilize the information or may not have received current information related to PA pressure measurement. On the contrary, nurses who had fewer years of experience may have current information but lack the clinical experience to utilize the information. In addition, it appears that there is inconsistent instruction related to the "basics" of PA pressure measurement as demonstrated back the low mean score in the Technical subset and the cognitive Level 1 subset.

Dolter (1987) asked nurses to rate their perceived ability with respect to various aspects of PA pressure measurement. A majority of the subjects rated themselves as confident to very confident of their abilities. However, Dolter did not relate perceived ability to test score. This study asked individuals to rate their perceived knowledge level related to PA pressure measurement. A majority of the nurses in the study rated themselves as at least competent. There was a difference in test scores based on an individual's perceived knowledge level of PA pressure measurement. Once again, there were individual variations within each demographic level. These data are of concern because, while most individuals perceived themselves as competent, test scores do not consistently demonstrate this ability. While subjective evaluation in many cases was consistent with an individual's abilities related to PA pressure measurement, objective analysis is required to validate this perception.

The cognitive subset scores were consistent with the knowledge deficits identified in the content subsets. The lowest cognitive subset score was Level 1: knowledge and comprehension (58.2%), while Level 2: application and analysis (70.8%), and Level 3: synthesis and evaluation (67%) were slightly higher. These findings were consistent with the item analysis that demonstrated a general knowledge deficit related terminology and translation of analog data into numerical values, with an increased ability to utilize hemodynamic data to plan and evaluate care.

The basic implication of this study is the need to use standardized, research-based information and terminology when instructing individuals on PA pressure measurement. It appears that critical care nurses' possess the ability to utilize hemodynamic data, but this ability is not supported by a firm understanding of the principles and technical aspects of hemodynamic monitoring. Therefore, it is important to evaluate an individual's knowledge base with regard to the basic principles of hemodynamic monitoring, such as interpreting an analog waveform, referencing, and controlling for respiratory variation at the knowledge and application level, before pursuing instruction regarding the clinical use of hemodynamic data or instruction at higher cognitive levels. Finally, this tool may be useful to evaluate individuals or small groups, and will allow for the development of specific instructional or orientation programs to meet their learning needs.

Limitations of the Study

The response rate of 18.1% required grouping of some levels within each demographic variable to facilitate data analysis, e.g., nurses with less than two years experience were grouped with nurses with two to three years of experience. A larger sample size would have allowed a more detailed evaluation of each of these levels. However, most demographic variables had an adequate number of subjects within each level to allow for statistical analysis.

The sample was taken from the membership of AACN. Membership in this professional organization may indicate more active involvement in professional development. Not all critical care nurses are members of AACN;, therefore, the sample was not representative of all critical care nurses. In addition, lack of knowledge may have led to some nurses to discard the questionnaire. Finally, the sample had more masters-prepared nurses, staff development coordinators, and CNSs than the AACN population. These individuals scored higher on all tests; therefore, the test scores for the target population may in fact be lower than reported.

There was some discrepancy among content experts with regard to cognitive level assignment for each item. Provision of operationalized definitions for each cognitive level and examples of items written for each cognitive level may have improved the congruence between experts. However, without knowledge of an individual's previous educational experiences related to PA pressure measurement, assignment of an item to one specific cognitive level is

difficult. In addition, there was a lack of congruence between the experts' evaluation of an item's relevance to a given content area. Once again, provision of more concise instructions for item scoring and the inclusion of items that were clearly irrelevant may have identified if a rater was having difficulty interpreting or following the instructions or whether the items were relevant to more than one area. Once again, provision of the operationalized definitions for each content area may have improved congruence among the experts.

A major limitation of the study surrounded the time required for the distribution of the tool. The questionnaire packet was mailed out at bulk rate, while the follow-up postcards were mailed first class. Five individuals notified the investigator that they had received the postcard but not the questionnaire packet. One individual was contacted, they indicated a questionnaire packet had beeen received one week after receipt of the postcard. All five of these individuals who indicated they had not received a packet were sent a second questionnaire packet via first class mail. Four of five returned the second questionnaire. There was no way to determine if other individuals failed to receive the questionnaire. In addition, because of the slow delivery rate, the time for analysis of the data was limited, with the subsequent result of a sample size that was smaller than desired.

Recommendations for Further Study

Development and utilization of this diagnostic tool represented the first step in the design of instructional interventions to

improve critical care nurses' knowledge and ability to utilize

information related to PA pressure measurement in the clinical

setting. Three nation-wide studies have identified consistent

knowledge deficits. Future research should be focused on evaluation

of the efficacy of various instructional methodologies to improve

this knowledge base. In addition, criterion validation of this tool

needs to be completed. This particular tool evaluated PA pressure

measurement in a patient with cardiac disease. While the basic

principles of hemodynamic monitoring do not change, alteration of the

scenario to evaluate this knowledge base with regard to other disease

processes, e.g., multisystem failure or sepsis, would provide further

guidance for educational programs. The items from this tool were

developed using an inference and action format. Further analysis of

this data could be conducted to evaluate congruency between inference

and action related to PA pressure measurement.

   To improve the response rate, a follow-up postcard that allowed

the subject to acknowledge receipt of the tool as well as their

decision whether to participate or not would provide the investigator

information regarding the actual receipt of the tool, and the

characteristics of the population who chose not to participate in the

study, and why they chose not to participate. In addition, the

questionnaire should be sent out first-class, or the follow-up

postcard should be sent approximately two to three weeks after

mailing of the questionnaire, to allow for delayed delivery of the

tool.

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APPENDIX A

Pulmonary Artery-Left Heart Pressure Relationship

List of Abbreviations

AL	anterolateral (myocardial infarction)
AS	antero-septal (myocardial infarction)
CVP	central venous pressure
HR	heart rate
I	inferior (myocardial infarction)
IP	inferior/posterior (myocardial infarction)
LA	left atrial
LAP	left atrial pressure
LV	left ventricular
LVDP	left ventricular diastolic pressure
LVEDP	left ventricular end-diastolic pressure
LVEDV	left ventricular end-diaitolic volume
MI	myocardial infarction
NS	nonsignificant
PA	pulmonary artery
PADP	pulmonary artery diastolic pressure
PAEDP	pulmonary artery end-diastolic pressure
PASP	pulmonary artery systolic pressure
PAWP	pulmonary artery wedge pressure
PEEP	positive end-expiratory pressure
PVR	pulmonary vascular resistance
PVW	pulmonary venous wedge
RA	right atrial
RAP	right atrial pressure
RV	right ventricular
V	variable

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Braunwald, Brockenbrough, & Ross (1961) V. LAP, LV pressure Sample: N = 18 patients studied due to presence of a heart murmur Normal RAP and PA pressure	Transeptal left heart catheterization Reference: 5 cm below sternal angle Simultaneous LAP and LV pressure measurements	**Pressures** Mean Range Mean LA 7.9 2-12 LA-RA 3.9 1-7 LA "a" onset 7.1 1-12 LA "a" peak 10.4 4-16 LA "a" wave 3.4 1-7 LA "z" point 7.6 1-13 LA "v" peak 12.8 6-21 LVEDP 8.7 5-12	No discussion of presence/absence of valvular disease Applicable only in patients with organic cardiovascular disease	Mean LA pressure and LA "z" point were similar to LVEDP
Kaltman, Herbert, Conroy, & Kossman (1966) V. LAP-PA-LVEDP difference	Right and left heart catheterization	56/70: *No PA-LVDP gradient* 14/70: LAP-LVDP gradient > 3 torr When PA < 17 torr: PA-LVEDP $r = 0.90$, $p < 0.001$ LAP-PADP (n = 46) $r = 0.87$, $p < 0.001$ HR effect NS	No individual data Limited to patients with normal PVR	In patients with congenital cardiac disorders, PADP, mean LAP, and LVEDP were highly correlated when pressures were less than 15 torr In patients with elevated left heart pressures, PA pressures were not good indices of left heart pressures Recommendations limited by lack of individual data

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Falicov & Resnekov (1970) V. PAEDP, LVEDP, PAWP, and PA "a" wave pressure Sample: N = 71: Positive murmur (n = 15); aortic valve stenosis (n = 15); aortic valve regurgitation (n = 15); mitral valve regurgitation (n = 12); myocardial infarction (n = 14). Exclusion: mitral valve obstruction, primary pulmonary vascular disease	Right and left heart catheterization Pressures averaged over entire respiratory cycle LVEDP: change in velocity of upstroke of pressure tracing ("z" point) PAEDP: lowest point on PA tracing PA "a" wave: presystolic wave in PA tracing	Normal LV function (n = 15) Range (torr) LVEDP 4-12 PAEDP 6-12 PAWP 4-12 LVEDP-PAEDP: r = 0.70, $p < 0.01$ PAEDP-PAEP : r = 0.69, $p < 0.01$ Maximum difference PAEDP-LVEDP: < 3 torr PAEDP-PAWP: < 3 torr LV dysfunction (n = 56) Range (torr) LVEDP 12-55 PAEDP 8-38 PAWP 9-37 LVEDP-PAEDP: r = 0.76, $p < 0.001$ PAEDP-PAWP: r = 0.92, $p < 0.001$ PA "a"-LVEDP: r = 0.94, $p < 0.001$* *in absence of increased PVR	Read over complete respiratory cycle Did not correlate LVEDP with PAWP Did not specify reference level or position	In the presence of LV dysfunction and elevated LVEDP, PAEDP correlated well with PAWP, but failed to accurately represent LVEDP In the absence of an elevated PVR, PA "a" wave was a good indicator of LVEDP in patients with LV dysfunction and normal sinus rhythm

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Forrester, Diamond, Ganz, Danzig, & Swan (1970) V: PADP, CVP, and LVEDP Sample: N = 23 critically ill patients; (n = 12) myocardial infarction	Pressures measured "at least" 30 minutes apart LVEDP measured in acute MI patients (n = 5)	No consistent relationship between CVP and LV filling pressures PADP > LVEDP by > 5 torr (30%) "Significant difference between LVEDP-PAWP (no stats) PAWP best correlated with mean LV diastolic pressure (no stats)	No statistics provided (abstract)	No recommendations can be made due to lack of specific data
Jenkins, Bradley, & Branthwaite (1970) V: PAEDP, LAP, PVR Sample: N = 28; (n = 4) chronic heart disease; (n = 18) post-op valve repair; (n = 2) acute MI; (n = 4) pulmonary embolism	Measured pressures before and after each intervention 1) Change atrial pressure 2) Inotropic therapy 3) Change atrial pressure and inotropic therapy Reference: sternal angle Separated into groups based on PVR: Group A: PVR < 2 units Group B: PVR 2.1-3.9 units Group C: PVR > 4 units	Difference LAP-PAEDP (torr) Range Mean A: 0.9-2.0 1.6 B: 2.1-3.9 2.8 C: 4.0-12.5 6.2 Correlation LAP-PAEDP A: $r = 0.95$, $p < 0.001$ B: $r = 0.87$, $p < 0.001$ C: $r = 0.76$, $p < 0.001$ No significant effect associated with HR	Small sample size for each disease state No discussion of disease effect on the pressure relationships	High correlation between LAP and PAEDP at all levels of PVR; however, large individual variations did occur with elevated PVR (group C) Use of PAEDP in patients with an elevated PVR is not recommended Small sample size limits generalizations and recommendations

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Bouchard, Gault, & Ross (1971) V: PA and LVEDP Sample: N = 50 patients undergoing diagnostic cardiac catheterization; (n = 24) normal LV function; (n = 26) LV dysfunction. Exclusion: mitral stenosis, increased PVR	LV and PA catheters Simultaneous PAEDP and LVEDP measurement Supine Analog data Intervention: 1) Acute increase in HR with LA pacing 2) Acute increase in LVEDP	Normal LV function LVEDP-PAEDP < 4 torr, $p < 0.2$ Abnormal LV function LVEDP > PAEDP (n = 20) Mean difference = 8 torr Normal PAEDP (< 12 torr) and increased LVEDP (mean 18.7) (n = 12) LV pre "a" wave-PAEDP: $r = 0.87$ (mean difference: 2.2 torr) Acute increase in HR (n = 14) Change LVEDP: -6 torr (-1 to -15 torr) Change PAEDP: +5 torr (1 to 13 torr) Acute increase in LVEDP Change LVEDP: +6 (2 to 11 torr) Change PAEDP: 0 (n = 6) Change PAEDP: +5 (n = 2)	Measured throughout respiratory cycle	With HR greater than 115 per minute, PAEDP did not reflect LVEDP In patients with an acute or chronic increase in LVEDP, atrial contraction augmented LVEDP, but this was not reflected on PA pressure tracing PAEDP failed to reflect an acute increase in LVEDP In patients with normal LV function, PAEDP was an accurate index of LVEDP

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Forsberg (1971) V: PA, LA, and LV pressures Sample: N = 158 patients with valvular or myocardial disease. Pulmonary dysfunction	Right and left heart catheterization Simultaneous measurement of PA, LA, and LV pressures Pressures averaged over ten heart cycles End-diastolic pressure measured at the "z" point,i.e.. foot of the "c" wave Supine	<u>LAP-LV pressure difference</u> Upstroke of LA "c" wave congruent with the upstroke of ventricular systole ("z" point) in patients with normal sinus rhythm and normal hemodynamics PADP-LAP = 1.4 torr PADP-LAEDP = -0.4 torr PADP equal to peak of LA "a" wave <u>Cardiovascular disease with PVR > 2 units</u> PAEDP-LAEDP-similar to patients without cardiovascular disease	Pressures averaged over 10 cardiac cycles Minimal statistical data	Recommendations limited by lack of specific data The LA "z" point was identical to LVEDP PADP was approximately equal to LVEDP In patients with cardiovascular disease, with normal PVR, PAEDP was similar to LAEDP (when compared with normal controls)

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Rao & Sissman (1971) V: Pulmonary venous and PA pressures Sample: N = 70 children; congenital heart disease; (n = 33); cardiac catheterization (n = 27); Normal PA pressures ; (n = 50); pulmonary hypertension (n = 20).	Right heart catheterization Impaction technique	Normal PA pressure PA-PWW difference (mean) PA-PWW systolic: 1.33 PA-PWW diastolic: 1.22 PA-PWW mean: 1.13 Maximum difference (torr) Systolic: 4 (11%) Diastolic: 7 (17%) Mean: 5 (15%) Pulmonary hypertension PA-PWW difference (torr) Systolic: 24.4 Diastolic: 7.4 Mean: 12.4	Did not specify characteristics of cardiac catheterization group	In patients with normal pulmonary vasculature, PA pressure was closely related to PWW pressure In patients with pulmonary hypertesnion, a large difference between PA and PWW pressures occurred In children with congenital heart disease, with increased PVR, use of PA pressure as an index of PWW pressure is not recommended
Balcon, Bennet, & Sowton (1972) V: PA and LV diastolic pressures at pre "a" point Sample: N = 15 with sample ischemic heart disease Excluded: pulmonary disease	Cardiac catheterization with right and left heart catheterization Pressures measured at the beginning of the upstroke of the "a" wave	LVEDP pre "a"- PAEDP pre "a"; r = 0.94, P < 0.001 PADP-LVEDP maximum difference: 5 torr (2.5%) 80% of the sample with < 2 torr difference	Did not correlate the LVEDP pre "a" with LVEDP Small sample size	In patients with an acute MI, PAEDP pre "a" was a good indicator of LVEDP pre "a" wave

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Rahimtoola & coworkers (1972) V: PA pressure, PAWP, LVDP Sample: N = 64 patients with an acute MI; n = 50 studied within three days of MI; n = 22 studied 3 to 12 weeks after acute MI	Right and left heart catheterization LVEDP "z" point: change in velocity of upstroke of LV tracing, after atrial contraction LVEDP pre "a" wave: prior to the increase in LVDP that occurs with atrial contraction PAEDP: lowest point on PA tracing Mean PA and PAWP: electronic integration Pressures averaged over 6 to 12 cardiac cycles	**Pressures** |	Mean	Range | LVEDP	19.6	6-42 LVDP pre "a"	11.9	0-27 LVEDP-LVDP pre "a" = 7.9 torr, $p < 0.001$ **Atrial contribution to LVEDP** LVEDP > 12 torr: 8.9 (2-18) LVEDP < 11 torr: 4.4 (1-11) **Pressure correlations** PAWP-LVDP pre "a"; $r = 0.90$ PAEDP-PAWP: $r = 0.77$# LVDP-PAWP: $r = 0.92$ NS with PVR < 2 units **Pressure differences** LVEDP-PAWP = 6.0, $p < 0.001$ PAWP-LVDP pre "a" = 0.8, NS PAEDP-PAWP (all) = 3.0, $p < 0.001$ PAEDP-PAWP (PVR < 2 units = 1.3, NS LVEDP-PAEDP = 4.7, $p < 0.001$ LVEDP-PA = 2.0, $p < 0.02$	Pressures averaged over 6 to 12 cardiac cycles	In patients with an acute MI, PA pressures did not accurately reflect LVEDP (due to atrial contraction) PAEDP failed to reflect PAWP in the presence of an increase in PVR LVDP pre "a" correlated well with PAWP; therefore, measurement of LV pressures (pre "a" and end-diastolic) will provide information about the potential for pulmonary edema and LV performance Discrepance between PAWP and LVEDP due to atrial contraction, i.e., "a" wave height

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Fisher, DeFelice, & Parisi (1973) V: LVEDP, PADP, PAMP, LVDP pre a", PAMP "a" wave Sample: N = 15; chest pain with coronary artery disease (n = 10); chest pain without coronary artery disease (n = 2); primary myocardial disease (n = 2); hypertension with mild aortic regurgitation (n =1)	Reference: 5 cm below sternal angle Supine Pressures averaged over 10 consecutive beats Mean pressures electronically integrated LVEDP: pressure read just prior to rapid systolic pressure rise - "z" point LV pre "a": distinct point between slow filling wave and atrial wave PAMP "a" peak of "a" wave Acute changes induced after left ventricular angiography	**Absolute value correlation** |	LVEDP	LV pre "a" | LVEDP	-	- | LV pre "a"**	-	- | PA*	0.88	0.78 | PADP*	0.87	0.80 | PAMP	0.98	0.93 | PAMP "a"	0.99	0.93 | *PADP and PA tended to underestimate LVEDP **Pressure differences > 5 torr** |	LVEDP	LV pre "a" | PA	40%	33% | PADP	40%	33% | PAMP	-	70% | PAMP "a"	3%	- | **Pressure relationship after sudden pressure change** |	LVEDP	LV pre "a" | LVEDP	1.0*	- | LV pre "a"	-	1.0* | PA	0.48	0.56 | PADP	0.63	0.78* | PAMP	0.76*	0.85* | PAMP "a"	0.87*	0.86* | *p < 0.01	No statistical evaluation of LVEDP and LV pre "a" differences	LVEDP and PAMP "a" most closeley correlated (absolute and change pressures) PAMP and PADP more closely correlated with LV pre "a", did not consistently reflect a change in LVEDP Large LV "a" wave led to a LVEDP-PAMP discrepancy, but a close relationship between LVEDP and PAMP "a" PADP and PAMP provided adequate indication of LV pre "a", and are useful indicators for the development of pulmonary edema

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Scheinman, Evans, Weiss, & Rapaport (1973)	Right and left heart catheterization	**Pressures (mean)** PAEDP 18.1 +/- -9.4 LVEDP 16.8 +/- 10.8	Small sample size in each subgroup	Difference between LVDP pre "a" and PAEDP not statistically significant, but clinically significant
V: PA and LV pressures	PAEDP: lowest point on PA pressure tracing	LVDP pre "a" 13.8 +/- 8.9	Did not specify amount of oxygen delivered	Atrial contraction did not
Sample: N = 25 patients in shock; MI (n = 12); Post-cardiac arrest (n = 3); Sepsis (n = 5); Hypovolemia (n = 3); Drug overdose (n = 1); Cerebrovascular accident (n = 1)	LVEDP: point immediately before rapid upstroke on LV pressure tracing	**Pressure correlations** PAEDP-LVEDP: r = 0.85s PAEDP-LVDP pre "a": r = 0.82s Change PAEDP-Change LVEDP: r = 0.81s (n = 27)	Did not separate effects of disease on pressure relationships	have a major effect on LV pressures Hypoxemia did not affect the high correlation between PAEDP and LVEDP
	LV pre "a": immediately before atrial contraction wave	Change PAEDP-Change LVDP pre "a": r = 0.85s (n = 22)	Excluded patients with increased PVR	PAEDP provided a good reflection of LVEDP in
Excluded: Severe bronchopulmonary or valvular heart disease	**Therapies** 1) High oxygen versus room air (n = 15) 2) Inotropic therapy (n = 7)	**Change in heart rate (r)** HR <100 >100 LVEDP-PAEDP 0.91 0.80 **Hypoxia vs oxygen therapy** NS effect on correlation between LVEDP-PAEDP **Inotropic therapy (r)** Before After LVEDP-PAEDP 0.92 0.86		patients in shock from a variety of causes; however, individual variations existed

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Watson & Kendall (1973) V: PAWP, LAP Sample: N = 700 patients with primary cardiac and valvular disorders	Right and left heart catheters Retrospective review of cardiac catheterization data	Total sample (N = 700) LAP-PAWP: $r = 0.93$ Disease specific LAP-PAWP (r) Coronary disease: 0.91 Mitral stenosis: 0.90 Mitral insufficiency: 0.93 Aortic stenosis: 0.91 Aortic insufficiency: 0.90 Mixed valvular disease: 0.93 PAWP < 25 torr NS difference LAP-PAWP PAWP > 25 torr PAWP - LAP difference significant ($p < 0.05$) PAWP < 10 torr LAP-PAWP difference +/- 2 torr	Pressures not all recorded simultaneously Limited discussion of individual variations	At PAWP < 10 torr, PAWP was predictive of LAP within +/- 2 torr While all correlations between PAWP and LAP were high, large individual variations in pressure did occur

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Calvin, Driedger, & Sibbald (1981) V: LVEDV, PAWP Variable levels of PEEP Sample: N = 57; Group 1: systemic sepsis (n = 24); Group 2: acute cardiac disease (n = 23) Excluded: Patients in shock or with valvular disorders	PA catheterization Radionuclide angiography for measurement of LVEDV Reference: midaxillary line End-expiration	<u>LVEDV-PAWP</u> Total: $r^2 = 0.01$, NS Cardiac: $r^2 = 0.03$, NS Sepsis: $r^2 = 0.02$ <u>No PEEP</u> Cardiac: $r^2 = 0.09$, NS Sepsis: $r^2 = 0.33$, $p < 0.01$ <u>PEEP</u> Cardiac: $r^2 = 0.08$, NS Sepsis: $r^2 = 0.21$, NS	Did not measure PVR	PAWP was a poor indicator of LVEDV in patients with sepsis or cardiac disease

Investigator Variables/Sample	Methods	Findings	Limitations	Implications

Saadjian, Cassol, & Torresani (1981)
 	
V: PAEDP, PAMP, LVDP, MI localization, PVR
 	
Sample: N = 127 with myocardial infarction. Excluded: right bundle branch block, ventricular septal defect, mitral regurgitation, chronic lung disease
	Simultaneous pressure recordings
	
	Right and left heart catheterization
	
	Studies 2 to 48 hours after onset of chest pain
	
	Reference: 5 cm below sternal angle
	
	LVEDP "z" point: change in velocity of upstroke of LV pressure tracing
	
	LV pre "a": just before atrial contraction
	
	LV "a" wave amplitude: measure from pre "a" wave pressure point
	
	LVEDP and PAEDP: measured at lowest point on tracing (averaged over 20 consecutive beats)
	
	PAMP: electronic integration
	PAMP systolic: top of the "a" wave
		Pressure correlations
		PAEDP-LVEDP
		Inferior MI (I) (n = 31)
		
		  Mean r
		PAEDP 13.0 -
		LVEDP 16.3 0.90
		LVDP 11.1 0.91
		LVDP pre "a" 9.9 0.92
		RA systolic 10.5 0.80
		PAMP 11.8 0.93
		
		Anteroseptal MI (AS) (n = 25)
		  Mean r
		PAEDP 12.2 -
		LVEDP 16.4 0.93
		LVDP 11.9 0.95
		LVDP pre "a" 9.3 0.95
		RA systolic 7.2 0.96
		PAMP 11.6 0.94
		
		Inferior/Posterior (IP) (n = 13)
		  Mean r
		PAEDP 11.4 -
		LVEDP 20.1 0.75
		LVDP 12.8 0.84
		LVDP pre "a" 11.9 0.81
		RA systolic 12.4 0.78
		PAMP 13.0 0.86
			LVDP-PAEDP measured over 20 consecutive cardiac cycles
			
			No specification of control for respiratory variation
				Location of the MI resulted in various pressure profiles that affect the correlation of right and left heart pressures
				
				Inferior-posterior infarcts were associated with a high degree of RV dysfunction, that was associated with a decrease in the correlation of PAEDP with LVEDP
				
				In patients with inferior-posterior MI, PAEDP should not be used as an indicator of left heart pressures

Investigator Variables/Sample	Methods	Findings	Limitations	Implications

Findings

Anterolateral MI (AL) (n = 40)

	Mean	r
PAEDP	19.2	-
LVEDP	25.9	0.88
LVDP	17.0	0.86
LVDP pre "a"	14.8	0.86
RA systolic	10.7	0.75
PAMP	17.7	0.89

Pressure Correlation

	LVEDP-PAMP	LVDP-PAMP
I	0.94	0.94
JP	0.87	0.89
AS	0.98	0.94
AL	0.92	0.85

Pressure Correlation

	LVEDP	LV pre "a"
LVEDP	1.00	-
LV pre "a"	-	1.00
PA	0.48	0.56
PADP	0.63	0.78
PAMP	0.76a	0.85a
PAMP "a"	0.87a	0.86a

$p < 0.01$

Non-significanct effect of heart rate and PVR on absolute and acute change values for all PA and LV pressures

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Levin & Glassman (1985) V. PAWP, LAP, PVR > 2.5 units Sample: N = 49 patients with chronically elevated PVR	Computerized evaluation of 10,000 cardiac catheterization files to identify patients with PVR > 2.5 units, and PAWP and LAP measurements	LAP-PAWP: $r = 0.84$, $p < 0.001$ with, PVR > 2.5 units NS correlation between HR, PASP, PADP, or cardiac output when PVR > 2.5 units	Retrospective Chronic elevation of PVR	Correlation between LAP and PAWP not altered by an elevation in PVR

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Lipp-Ziff & Kawanishi (1991)	Right/left heart catheterization	**Pressure correlations with**	Normal PVR	In patients with cardiac disease with normal sinus rhythm and normal PVR, PAD 0.08 after onset of QRS had best correlation with LVEDP
		LVEDP		
V: PADP, PADP 0.04, PADP 0.08, LVEDP	PAD: last small rounded portion of the waveform in diastole, commonly the lowest point	All (n = 100)	No mechanical ventilation	
		r SEE		
		PAD 0.82 4.43		
		PAD 0.04 0.86 3.96		
		PAD 0.08 0.88 3.61		
Sample: N = 100; stable CAD (n = 44); unstable angina (n = 37) conduction defects (n = 23); other cardiac disease (n = 19)	PAD 0.04: 0),04 sec after onset of QRS	CAD (n = 44)	Small sample size in some subsets	Individual variations needs to be assessed
		r SEE		
		PAD 0.79 4.49	Did not compare PAD with PAWP	
	PAD 0.08: 0.08 sec after onset of QRS	PAD 0.04 0.84 3.99		
		PAD 0.08 0.86 3.74		
Excluded: Pulmonary disease and mitral valve disease, HR > 130, mechanical ventilation	LVEDP: peak of R wave	Unstable angina (n = 37)		
		r SEE		
		PAD 0.87 3.56		
		PAD 0.04 0.88 3.88		
	End-expiration	PAD 0.08 0.94 2.46		
		Normal sinus rhythm (n = 69)		
	Measurements all taken from one waveform	r SEE		
		PAD 0.85 3.97		
		PAD 0.04 0.89 3.47		
	Reference: Midanteroposterior chest level	PAD 0.08 0.89 3.45		
		Bradycardia (n = 22)		
		r SEE		
		PAD 0.76 4.48		
		PAD 0.04 0.84 3.77		
		PAD 0.08 0.92 2.68		

Investigator
Variables/Sample   Methods   Findings   Limitations   Implications

Heart Block (n = 23)

	r	SEE
PAD	0.75	5.14
PAD 0.04	0.86	3.96
PAD 0.08	-0.90	3.37

Other cardiac disease (n = 19)

	r	SEE
PAD	0.81	5.69
PAD 0.04	0.87	4.79
PAD 0.08	0.85	5.12

Pressure Differences (mean)

LVEDP	18.6	-------
PAD	15.7	$p < 0.05$
PAD 0.04	16.3	$p < 0.05$
PAD 0.08	17.1	$p > 0.05$

Appendix B

Effect of Mechanical Ventilation on the Accuracy
of Pulmonary Artery Pressure Measurement

<u>List of Abbreviations</u>

ABG	arterial blood gas
ICU	intensive care unit
IPPV	intermittent positive pressure ventilation
LAP	left atrial pressure
LV	left ventricular
LVEDP	left ventricular end-diastolic pressure
MV	mechanical ventilation
NS	nonsignificant
O2	oxygen
PA	pulmonary artery
PADP	pulmonary artery diastolic
PAMP	pulmonary artery mean
PaO2	partial pressure oxygen, arterial
PASP	pulmonary artery systolic
PAWP	pulmonary artery wedge pressure
PEEP	postive end-expiratory pressure
RAP	right atrial
V	variables

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Davison, Parker, & Harrison (1978) V: On/Off MV, with/without PEEP ($<$ 10 cm H_2O) PAWP Sample: N = 29 ICU patients on MV; PEEP $<$ 10 cm $H2O$ (n = 13)	Measured lowest PAWP during last end-expiratory period before MV disconnection Lowest PAWP off MV Highest PAWP during recording (on or off MV) End-expiration Analog data Reference: 10 cm from the dorsal surface	IPPV without PEEP (n = 16) **Mean PAWP** On: 10.3 torr Off: 10.3 torr IPPV with PEEP (n = 13) **Mean PAWP** On: 12.5 torr Off: 11.7 torr	Did not reference to the phlebostatic axis Only examined PAWP Mean data only	PAWP at end-expiration on/off MV were consistent in patients with PEEP ($<$ 10 cm H_2O) and without PEEP

Investigator/ Variables/Sample	Methods	Findings	Limitations	Implications
Shinn, Woods, & Huseby (1979) V: On/off MV (no PEEP); PA pressures Sample: *N* = 18 critically ill patients on MV	PA pressures on and off MV Flat, supine Pressures measured over one typical respiratory pattern Reference: Phlebostatic axis :	Mean difference on/off MV PASP: -1.1 (p < 0.05) PADP 1.6 (p < 0.001) PAMP: -1.5 (p < 0.001) PAWP: -0.3 (p < 0.4) Individual differences on vs off MV 72/144 pressures < 2.5 torr 4/72 > 4 torr (5.6%) Mean difference PADP-PAWP On: -5.2 (p < 0.01) Off: -3.9 (p < 0.05)	No randomization of on/off sequence All patients supine, flat Data read through an entire respiratory cycle on/off MV	Individual changes were small; therefore, the patient may be left on MV for measurement of PA pressures. Individual responses need to be assessed. PADP should not be used in place of PAWP as an index on LVEDP in patients on MV
Grose & Woods (1981) V: On/off MV (no PEEP); Backrest elevation change; PA pressures Sample: *N* = 60 acutely ill patients	Repeated measures on/off MV Supine backrest elevation change: Group A: 0-20-0 degrees Group B: 20-0-20 degrees Reference: Phlebostatic axis	Mean difference on/off MV PASP: -1 (p < 0.01) PADP -2 (p < 0.01) PAMP: -1 (p < 0.01) PAWP: -1 (p < 0.01) Individual differences 13/60 had differences on/off MV that exceeded expected fluctuation in pressures (confirmed by repeated measures)	No patients on PEEP Data not read at end-expiration	Most of the patients had small differences on/off MV, but clinically significant variations did occur. Pressures should be evaluated on and off MV to assess for individual variations

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Van Sciver (1982) V: On/off MV (PEEP = 5 cm H_2O); PA pressures; Spontaneous and controlled ventilation Sample: $N = 14$ ICU patients on MV; on PEEP ($n = 11$); LV dysfunction ($n = 12$)	Measured PA pressures on/off MV Analog data End-expiration Spontaneous and controlled ventilation	Mean difference on/off MV PASP: $p < 0.001$ PADP: $p < 0.001$ PAWP: $p < 0.05$ Significant changes in all patients with LV dysfunction. No significant differences in patients without LV dysfunction ($n = 2$)	No individual or group data presented Did not specify reference point, or length of time off MV before readings taken	Recommendations limited without individual data Higher PA/PAWP can be expected in patients with LV dysfunction when removed from MV
Gershan (1983) V: On/off MV (PEEP 0-20 cm H_2O); PA pressures Sample: $N = 30$ ICU patients on MV with PEEP	Measured PA pressures on/off MV with digital and analog data End-expiration Pressures measured 10 seconds off MV PEEP not varied Reference: Phlebostatic axis Supine	PAWP signficantly decreased off MV ($p < 0.01$) PASP and PADP increased off MV with PEEP of 16-20 cm H_2O PASP and PADP decreased off MV with PEEP < 15 cm H_2O	No validation of PA catheter position Did not vary the level of PEEP No individual data Did not address effect of removal of MV on O_2 delivery	Without individual data no recommendations can be made

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Lookinland (1989)	On/off MV	**Mean difference on/off MV**	Normal cardiac and pulmonary function	In patients receiving low level PEEP (< 10 cm H_2O), with adequate blood volume, PA pressures on MV are reliable
V: On/off MV (PEEP < 10 cm H_2O); hypovolemic, normovolemic, or hypervolemic; O_2 delivery; RA and PA pressures	Blood volume determined by tagged albumin	PAMP: +0.3 (NS) PASP: +0.3 (NS) PADP: +0.1 (NS) PAMP: +0.5 (NS) RAP: +0.5 (NS)	Small sample (n = 3 with blood volume deficit > 1 liter)	Removal from MV results in hypoxemia; therefore, removal from MV should be performed cautiously
	ABG's at 0, 15, 30, 42, and 52 seconds off MV, and a 5, 15, 30, and 60 minutes after return to MV.	**Individual differences** Three patients with blood volume deficits > 1 liter demonstrated a "significant" decrease in PA pressures off MV (no stats)	Low level PEEP Limited statistical analysis of data	
Sample: N = 30 critically ill patients (mean age 45)	Flat or 20 degrees backrest elevation			
	Analog data	"Significant" decrease in PaO_2 for 30 minutes after return to MV (p < 0.05)		
	Reference: Phlebostatic axis			
	End-expiration over two respiratory cycles			

APPENDIX C

Effect of Positive End-Expiratory Pressure on
Pulmonary Artery Pressure Measurement Accuracy

List of Abbreviations

BP	blood pressure
CXR	chest radiograph
LA	left atrial
LAP	left atrial pressure
MV	mechanical ventilation
NS	nonsignificant
PA	pulmonary artery
PAM	pulmonary artery mean
PAWP	pulmonary artery wedge pressure
PEEP	positive end-expiratory pressure
RA	right atrial pressure
RL	ringers lactate
V	variables

Investigator

Variables/Sample	Methods	Findings	Limitations	Implications
Lozman (1974)	Part 1	PAWP-LAP correlation	Small sample	Despite small mean difference in PAWP-LAP, large individual variations did occur above 5 cm H_2O PEEP
V: Altered levels of PEEP at 0, 5, 10, 15, 10, 5, 0; LAP, PAWP, PAWP-LAP difference	Sequential increase in PEEP in 5 cm H_2O increments	In 9/32 data sets (PEEP > 10 cm H_2O) PAWP did not reflect changes in LAP	No statistical evaluation of PAWP-LAP difference	
	End-expiration	At < 10 cm H2O PEEP, PAWP was a good reflection of LAP. Individual differences: -16 to +6 torr (mean 0.38) Correlation: $r = 0.11$, NS	Did not use phlebostatic axis	PAWP was a good indicator of LAP at/below 5 cm H_2O PEEP
Sample: $N = 5$ cardiac surgery patients on MV with PEEP	Reference: 5 cm below angle of Louis at the sternum			
	LA and PA catheters	At/below 5 cm H_2O PEEP PAWP-LAP significantly correlated ($r = 0.83$, $P < 0.01$)	No information on verification of catheter placement	A possible cause of the discrepancy between PAWP and LAP was unequal balloon inflation, resulting in occlusion of the catheter tip against the vessel wall
	Part 2	Part 2		
	In vivo/vitro dog study ($n = 2$) of morphology of PA catheter	In vitro: 5 of 6 balloon inflations preferentially inflated to one side, resulting in occlusion of the catheter tip against the wall of the vessel		
		In vivo: ($n = 1$) catheter tip bulged against the vessel wall		

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Hobelmann, Smith, Virgilio, Shapiro, & Peters (1974) V: PEEP at 0-5-10-15-20 cm H2O; PAWP PAWP, LAP, RAP Sample: N = 6 baboons on MV with PEEP	Vascular pressures measured during control and at end of 20 minutes of PEEP at 5,10,15,20 cm H2O	Mean difference PAWP-LAP 0 cm: +1.0 5 cm: +1.0 10 cm: +2.5 15 cm: +6.5 20 cm: +9.5 Mean difference from control PEEP 5 10 15 20 PAWP 3.0 9.0 17 21 PAWP 0.5 3.5 7.0 11 LAP 0.5 2.0 1.5 1.5 RAP 1.0 1.5 2.5 3.0	Healthy baboon model No individual data Nonrandomized application of PEEP No repeated measures	Mean data would indicate that PAWP was not a good index of LAP above 10 cm H2O PEEP; however without individual data or statistical analysis recommendations cannot be made
Roy, Powers, Feustel, & Dutton (1977) V: PEEP at 0,5,10, and 15 cm H2O; Open vs closed thorax; Catheters positioned above or below left atrium; PAWP-LAP difference Sample: N = 10 healthy dogs	Randomized application of PEEP at 0,5,10, and 15 cm H2O End-expiration Verification with lateral radiograph Stabilization period: 2 minutes at each PEEP level	No significant differences between open/closed thorax Mean PAWP-LAP difference PEEP Above Below 0 0.6 -0.5 5 1.0 -0.4 10 3.2 0.7 15 6.3 1.8	Did not specify reference level, when pressures read during ventilatory cycle, or method of catheter position verification Small sample size Healthy dog model No repeated measures No individual data	Catheter placement below the left atrium (zone 3) will increase reliability of PAWP measurements up to 15 cm H2O PEEP, provided airway pressure is less than LAP plus the vertical distance the catheter is placed below the left atrium Recommendations are limited by the lack of individual data, and the healthy nonhuman model

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Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Zarins, Virgilio, Smith, & Peters (1977) V: PEEP in 5 cm H_2O increments from 0-20-0 cm H_2O; Volume status: acute hypervolemia, normovolemia, chronic fluid overload, and functional hypovolemia; PA and LA pressures Sample: N = 6 healthy baboons	Altered PEEP level from 0-20-0 in 5 cm H_2O increments Fluid volume status altered End-expiration Analog data	PAWP-LAP correlation Normovolemia: ,PAWP no longer reflected LAP at 20 cm H_2O ($p < 0.05$) Acute Hypervolemia PAWP = LAP at all levels of PEEP Chronic fluid overload PAWP = LAP at all levels of PEEP Functional hypovolemia At PEEP = 5 cm H_2O all baboons had a significant decrease in cardiac output. 4 of 5 baboons died during functional hypovolemia study Repeated measures during normovolemia demonstrated a mean change of 4 torr for PAWP and 5 torr for LAP	Healthy baboon model Small sample No individual data	Fluid therapy with elevation of LAP above alveolar pressure overcomes the pulmonary vascular compression, and equalizes PAWP and LAP During acute hypervolemia PAWP and LAP were elevated above 20 cm H_2O, potentiating clinical compromise. The beneficial stabilizing effect of fluid therapy must be weighted against this clinical risk

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Kane, Askanzi, Neville, Mon, Hason, & Webb (1978) V: PEEP 5-15 cm H_2O in 5 cm increments; PA catheter position above/below left atrium; Normovolemia, hypervolemia, and hypovolemia; PAWP-LAP difference Sample: $N = 19$ healthy dogs	Sequential elevation of PEEP: 5-10-15 cm H_2O Group 1: PA catheter 4 cm above left atrium ($n = 8$) Group 2: PA catheter 4 cm below left atrium ($n = 8$) Group 3: PA catheter above left atrium. LAP increased with infusion of RL and dextrose, and lowered with bleeding (BP = 40 torr) Analog data Reference: level of the left atrium	Mean PAWP-LAP relative to left atrium PEEP 5 10 15 Above 1.6 6.5 10.6ª Below 0.8 -1.1 0.2 ªp < 0.05 Mean PAWP-LAP depending on volume status PEEP 5 10 Normovolemia 2.2 7.0 Hypervolemia -0.7 0.4 Hypovolemia 4.3 9.4	Small sample Healthy dog model No individual data No discussion regarding when pressures read during respiratory cycle	Without individual data recommendations are limited In the presence of either increased airway pressure or low LAP, PAWP may not serve as a reliable indicator of LAP

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Tooker, Huseby, & Butler (1978) V: RAndom PEEP from 0-30 cm H₂0, in 5 cm increments; PA catheter position; Normal vs injured lung; PAWP-LAP difference Sample: *N* = 9 dogs	Random PEEP application from 0-30 cm H20 Two catheters; positoned above and below the left atrium Pulmonary edema induced by oleic acid (*n* = 6); normal (*n* = 9) End-expiration Lateral chest radiograph Reference: left atrium (atmospheric)	Mean PAWP-LAP difference Normal lung PAWP (lower) highly correlated with LAP throughout entire range of PEEP (r = 0.96) PAWP (upper) greater than LAP at all level of PEEP (r = 0.73) Pulmonary edema PAWP (lower) correlated highly with LAP (r = 0.97); PAWP (upper) demonstrated wide scatter (r = 0.65)	No individual data No repeated measures Did not specify length of time for stabilization, total study time, or method of controlling for respiratory variation Small sample Dog model	Without individual data and noted limitations, recommendations are limited Study demonstrated that catheter placement below the left atrium increased the reliability of PAWP as an indicator of LAP across a wide range of applied PEEP in normal and diseased lungs

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Berryhill and Benumof (1979) V: Sequential PEEP from 0-20 cm H$_2$O; Controlled vs spontaneous ventilation; Catheter position above or below left atrium; Normal vs damaged lung; PAWP-LAP difference Sample: N = 7 dogs	PAWP and LAP from catheter in up or down lung at 0-20 cm H$_2$O PEEP during controlled and spontaneous ventilation Lung damaged with oleic acid and pressures repeated Two catheters placed: above or below left atium	No change in transmural LAP at any level of PEEP <u>Controlled ventilation</u> Above: PAWP > LAP at 5 cm H20 PEEP ($p < 0.01$) At PEEP > 10 cm H$_2$O PAWP changes followed PEEP <u>Down: NS differences</u> <u>Spontaneous Ventilation</u> NS difference at all levels of PEEP <u>Spontaneous Ventilation</u> <u>Compliant vs noncompliant</u> NS change in LAP in either lung at any level of PEEP <u>Controlled Ventilation</u> <u>Compliant lung:</u> Up: PAWP > LAP at PEEP = 5 torr ($p < 0.01$) Down: PAWP > LAP at PEEP = 15 torr ($p < 0.01$) <u>Noncompliant lung:</u> Up: PAWP > LAP at PEEP = 10 torr ($p < 0.01$) Down: PAWP > LAP at PEEP = 15 torr ($p < 0.01$)	No individual data No randomization of variables No repeated measures Did not specify length of time for protocol or stabilization period	Recommendations limited by lack of individual data Spontaneous ventilation afforded complete protection of the accuracy of PAWP as an indicator of LAP Noncompliant lung provided moderate protection of the accuracy of PAWP as a reflection of LAP

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Shasby & coworkers (1981) V: PEEP from 0-15 cm H₂0; Catheter position above or below left atrium; LAP (n = 12); PAWP-LAP difference Sample: N = 30 cardiac surgery patients	PEEP incrementally applied for 0-15 cm H₂0 Catheter position determined by lateral chest radiograph End-expiration pressures averaged over three respiratory cycles Analog data	43% of the catheters wedged 1 cm above LA marker PAWP-LAP difference Above/LAP < 5 torr: < 1 torr at 0 torr; PEEP; 13.5 torr at 13 torr PEEP Above/LAP > 5 torr: Significant difference at 11 torr (p < 0.05) Below: No significant difference at any level of PEEP (p > 0.2)	Did not specify length of time for stabilization, reason for cardiac surgery, or reference level No individual data Limited statistical analysis of data	Recommendations limited by lack of specific data When catheter was above the left atrium, with LAP < 5 torr, a large difference between PAWP and LAP occurred When catheter tip was above the left atrium with LAP > 5 torr, small but statistically significant differences between PAWP and LAP occurred with increased PEEP Positioning the catheter below the left atrium increased the reliability of the PAWP as an indicator of LAP

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Malanga, Hasan, Braman, Carrao, & Most (1984) V: PEEP 0-30 cm H_2O; catheter position above or below the left atrium; PAWP-LAP difference Sample: $N = 10$ pigs	Catheters (2) placed into right and left PA	PAWP-LAP difference Above: PAWP >.LAP at > 10 cm H_2O PEEP $(p < 0.001)$. Below: No significant difference up to 20 cm H_2O PEEP	No individual data No information provided about method of data collection (analog vs digital), stabilization period, length of time for protocol, or method for controlling respiratory variation Healthy pig model	Recommendations limited due to noted limitations Positioning the animal in the lateral position, with the resultant placement of the catheter in the dependent position increased the reliability of PAWP as an indicator of LAP

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Hasan, Malanga, Braman, Carrao, & Most (1984) V: PEEP at 0-30 cm H_2O; Right and left lung PA catheters; PAWP-LAP difference Sample: $N = 10$ pigs	Simultaneous LAP and PAWP from right and left PA catheters in right or left lateral position PEEP randomly altered in 5 cm increments from 0-30 cm H_2O Random position change Analog data End-expiratory pressure averaged over three respiratory cycles Stabilization period: 2 to 3 minutes	17/20 catheters positioned below the left atrium PAWP-LAP difference Above: Significant difference in PAWP-LAP at all levels of PEEP Below: Significant difference in PAWP-LAP at PEEP = 20 cm H_2O, mean = +3.0 ($p < 0.05$), and at 30 cm H_2O PEEP mean = 7.8, ($p < 0.05$) Lower catheter correlated with LAP over 0-15 cm H_2O ($r = 0.82$). Upper catheter overestimated LAP at PEEP = 0-20 cm H2O ($r = 0.64$) Placement of above catheter into dependent position by right or left lateral position cahnge resulted in increased accuracy of LAP measurement over 5-20 cm H_2O PEEP range ($p < 0.05$)	Healthy pig model No repeated measures No individual data	Recommendations limited by lack of individual data PAWP measured in the lateral position, when the catheter tip is vertically positioned below the left atrium, more accurately reflected LAP over the usual range of PEEP, than did PAWP measured in the supine position when the catheter was at or above the left atrium level

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Hasan, Weiss, Braman, & Hoppin (1985) V: PEEP from 0-22 torr; Normal vs injured lung; Right and left PA catheters; Right and left PAWP; PAWP-LAP difference Sample: N = 12 dogs	Random application of PEEP from 0-22 torr Oleic acid induced lung injury (n = 10) Unilateral lung injury (n = 7) End-expiration Reference: midaxillary line	16/20 catheters below left atrium Significant increase in PAWP-LAP difference at > 15 torr in normal and bilaterally injured lungs ($p < 0.05$) <u>Unilateral lung injury</u> Normal: Significant difference in PAWP-LAP at > 7 torr ($p < 0.05$) Injured: Significant difference in PAWP-LAP at 15 torr ($p < 0.05$) <u>Position effect</u> Normal lung dependent Increased PAWP-LAP correlation up to PEEP = 11 torr (no stats) Injured lung elevated PAWP-LAP difference increased at PEEP > 15 torr	Did not specify method of data collection (analog vs digital)	Lung injury may protect the pulmonary vasculature from the collapsing effect of increasing alveolar pressure PAWP obtained from catheter in injured lung more accurately reflected LAP than simultaneous PAWP from normal lung At PEEP < 7 torr, PAWP-LAP difference from a catheter inserted in a normal lung were corrected by the use of lateral position (placement of the catheter into a dependent position)

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Rajacich, Burchard, Hasan, & Singh (1989) V: PEEP 0-20 cm H_2O; PA catheter position above or below left atrium; Supine or lateral position; PA pressures; PAWP-LAP difference Sample: $N = 17$ cardiac surgery patients	Sequential increase in PEEP from 0-20 cm H_2O in 5 cm increments Analog data End-expiration Reference: Supine: Midaxillary line Lateral: Transducer adjusted to obtain baseline LAP measured in the supine position Positioned in right or left lateral position to place the PA catheter above or below the left atrium	16/17 catheters in right PA. One catheter above the left atrium Supine ($n = 17$) All pressures significantly different than baseline at PEEP > 15 cm H_2O ($p < 0.01$) Tip down lateral ($n = 12$) No significant difference between PAWP-LAP at any level of PEEP Tip up lateral ($n = 5$) Significant difference between PAWP-LAP at PEEP > 15 cm H_2O	No individual data No specification of degree of lateral turn No repeated measures Normal cardiac and pulmonary function 16/17 catheters in right lung (questionable effect of position change)	Recommendations are limited by lack of specific methodological information Tip down position may increase the reliability of PAWP measurement as an indicator of LAP in cardiac surgery patients

APPENDIX D

Effect of Supine Position Change on

Pulmonary Pressure Measurement Accuracy

List of Abbreviations

AP	anterior-posterior
CABG	coronary artery bypass graft
HOB	head of bed
ICS	intercostal space
ICU	intensive care unit
LAP	left atrial pressure
MI	myocardial infarction
MV	mechanical ventilation
MVR	mitral valve replacement
NS	nonsignificant
PA	pulmonary artery
PADP	pulmonary artery diastolic
PAMP	pulmonary artery mean
PASP	pulmonary artery systolic
PAWP	pulmonary artery wedge pressure
PEEP	positive end-expiratory pressure
V	variables

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Prakash, Parmley, Dikshit, Forrester, & Swan (1973) V: 0 and 70 degrees supine backrest elevation; PA pressures Sample: N = 21 s/p MI, without cardiogenic shock or pulmonary edema	Backrest elevated from 0 to 70 degrees, with legs horizontal Reference: 5 cm vertically below sternal angle, 4th ICS Stabilization: Five minutes after each position change	**Mean pressure changes** PASP: +2.3 (p < 0.01) (n = 17) PAMP: +3.4 (p < 0.01) (n = 20) RAP: +2.9 (p < 0.001) **Individual differences** PASP: -3 to +8 torr PAMP: -4 to +18 torr RAP: 0 to +5 torr	Backrest sequence not varied PADP not measured Phlebostatic axis not used as reference level Small, homogeneous sample Did not specify when pressures read during the ventilatory cycle	Limited due to large individual variations Authors concluded that patients can be positioned for comfort (not supported by data)
Hansen (1978) V: 0, 20, 45 degrees supine backrest elevation; PA pressures Sample: N = 18 ICU patients	Backrest elevation changed sequentially 0, 20, 45 degrees Analog waveform except if respiratory variation occurred Pressure recorded over one respiratory cycle Reference: Phlebostatic axis Stabilization: 5 minutes	10/18 patients with variations in pressures greater than 4 torr **Mean differences** **0-20 degrees** PASP: -0.6 (NS) PADP: -0.3 (p < 0.05) PAMP: -0.9 (NS) PAWP: -1.6 (NS) **0-45 degrees** PASP: -0.8 (NS) PADP: +1.1 (p < 0.05) PAMP: -0.3 (NS) PAMP: -0.7 (NS)	Not read at end-expiration Small sample Did not vary backrest sequence No repeated measures Total data collection time: 30 to 60 minutes	Placing the patient in a position other than supine for PA pressure measurements not recommended based on this study Individual differences may have been r/t hemodynamic changes over time, and not due to position change

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Woods & Mansfield (1976) V: 0,.20,45, 90 degrees, and dangle; PA pressures Sample: $N = 10$ stable cardiac patients during exercise testing	Backrest elevation changed sequentially 0,20,45,90 degrees and dangle Stabilization period: 15 seconds to 4 minutes (mean 2.1 min) Pressures averaged over one respiratory cycle Reference: phlebostatic axis	Largest mean change PASP: -3.4 ($p < 0.05$) PADP: +1.6 ($p > 0.05$) PAMP: -1.6 ($p > 0.05$) PAWP: -1.2 ($p > 0.05$) Individual differences 0-20-45 degrees PASP: 1-11, mean 4.0 PADP: 1-8, mean 2.9 PAMP: 1-12, mean 4.0 PAWP: 1-3, mean 1.9	Read throughout entire respiratory cycle Backrest sequence not varied No repeated measures All subjects with normal PA pressures	Findings suggest effect of position change may be small for most individuals, but these findings can not be generalized beyond the sample
Burrage (1979) V: Supine backrest elevation at 0,20, and 45 degrees; PA pressures Sample: $N = 30$ ICU patients	Backrest elevation changed sequentially 0,20,45 degrees Reference: phlebostatic axis Analog data Waveform interpreted throughout the respiratory cycle Stabilization period: 5 to 24 minutes	Mean differences not significant ($p > 0.05$) for all PA pressures at all backrest elevations PAWP range (mean) 0-20: -4 to +5 (-0.241) 0-45: -7 to +6 (0.172) PAWP change greater than 4 torr during position change ($n = 4$)	Sequence not varied No repeated measures Pressures not read at end-expiration No analysis of individual variations Variable stabilization period Nonrandomized position change	Based on small mean changes it appears that patients can be placed at backrest angles of 20 to 45 degrees for reproducible pressure measurements. However, individual variations need to be assessed

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Fournier, Mensche-Dechene, Ranson-Bitker, Valladares, & Lockhart (1979)	PA catheter position	Mean pressure differences	Small sample size	Limited recommendations based on lack of mean data
	Position change from supine to sitting with legs dangling	PASP: -8 (p < 0.025) PADP: -4 (p < 0.005) PAMP: -4 (p < 0.005) PAWP: -6.5 (p < 0.025)	Mean data only	
V: Position change from supine to sitting; mean PA pressures	Pressures averaged over three respiratory cycles		Pressures averaged electronically	Based on mean data, placement of the patient in a sitting position for PA pressure measurements is not recommended
Sample: N = 8 patients with unilateral lung carcinoma	Mean pressures measured electronically		Pressures averaged over three respiratory cycles	
Laulive (1982)	Sequential elevation of backrest at 0,20,45, 60, and 0 degrees	Mean differences (all degrees)	Pressures read from scale on oscilloscope	It appears that reproducible PA measurements can be obtained in cardiac ICU patients with backrest elevations to 60 degrees
V: Supine backrest elevation of 0,20, 45,60,0 degrees; PA pressures	Length of data collection time not specified	PASP: 0.28 (NS) PADP: 1.03 (NS) PAWP: 0.57 (NS)	Interrater reliability completed randomly with an available staff nurse	
Sample: N = 30 cardiac ICU patients	End-expiration	Individual variations all 2 torr or less	Did not specify stabilization time	Individual variations must be assessed
	Reference: 4th ICS/ midaxillary line		Did not specify total time for study	

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Miller & Chulay (1982) V: Supine backrest elevation at 0,20,30, 45,0 degrees; mean PA pressures Sample: N = 15 cardiac surgery patients; CABG (n = 14), MVR (n = 1)	Sequential elevation of backrest at 0,20, 30,45,0 degrees End-expiration Phlebostatic axis	No significant difference in mean readings when the angle of the backrest was changed Mean differences < 1 torr for all subjects	No individual data Did not specify method of data collection (analog vs digital) Did not specify stabilization period or length of time for study	Mean differences were small, but without discussion of individual changes, no change in practice can be recommended
Woods, Gross, & Laurent-Bopp (1982) V: Supine backrest elevation of 0-20-0 degrees or 20-0-20 degrees; PA pressures Sample: N = 126 ICU patients (n = 60 on MV without PEEP)	Sequential elevation of backrest elevation Group A: 0-20-0 Group B: 20-0-20 Stabilization period: 5 minutes Total time: 30 minutes Reference: Phlebostatic axis Analog data If respiratory variation occurred, PASP and PADP averaged over one respiratory cycle	Combine group ranges PASP: 2-7 (mean 3.9) PADP: 1-6 (mean 3.4) PAMP: 1-5 (mean 2.8) PAMP: 0-7 (mean 3.4) 0-20-0 (group A) PAMP and PAMP decreased 0.3 to 2.4 torr ($p < 0.01$) All other changes NS 20-0-20 (Group B) Mean changes NS 4/126 changes exceeded normal fluctuation	HOB elevation only to 20 degrees Pressures read over one respiratory cycle	Unless clinically significant differences are found in individual patients, reliable pressures can be obtained at backrest elevation angles of 20 degrees or less, without repositioning the patient flat

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Chulay & Miller (1984) V: Supine backrest elevation at 0-20-30-45-0 degrees; mean PA pressures Sample: *N* = 32 cardiac surgery patients *n* = 10 on MV with 5 cm PEEP	Sequential change of backrest at 0,20,30,45,0 degrees End-expiration Total time: 10 minutes	**Mean pressure differences** All mean pressures < 1.3 torr compared with control at all backrest elevations **Individual differences** All differences < 1.4 torr No statistically or clinically significant changes	No individual data presented except in discussion No specification of stabilization period No specification of reference level	PA pressures may be taken reliably up to 45 degrees supine backrest elevation
Clochesy, Hinshaw, & Otto (1984) V: Supine backrest elevation at 0,20, 45, and 60 degrees; mean PA pressures Sample: *N* = 17 ICU patients on MV with PEEP ≥ 5 cm H$_2$O	Randomized change in backrest at 0,20, 45, and 60 degrees Phlebostatic axis End-expiration Stabilization period: 5 minutes	**Range of mean differences** PASP: -0.5 to +0.5 (NS) PADP: +0.1 to +0.4 (NS) PAMP: +0.4 to +1.6 (NS)	No individual data presented No discussion of method for data interpretation (digital versus graphic)	Mean differences were small, but without a discussion of individual changes, a change in practice is not recommended

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Retailliau, McGregor-Leding, & Woods (1985) V: Supine backrest elevation at 0-30-0 or 30-0-30 degrees; LAP Sample: N = 34 cardiac surgery patients; n = 28 on MV with 3-5 cm H$_2$O PEEP	Sequential alteration in supine backrest elevation Group A: 0-30-0 degrees Group B: 30-0-30 degrees Stabilization period: 5 minutes Analog data End-expiration Phlebostatic axis	Mean LAP difference 0-3 torr: 26/34 (81%) > 4 torr: 6/34 (16%) (4/6 with left ventricular dysfunction or mitral valve disease) Mean LAP difference between 0-30 degrees was statistically significant ($p < 0.001$) LAP fluctuation over 30 minutes: 0 to 3 torr	18 patients received colloids or blood during the study period	LAP can be reliably measured up to 30-degrees supine bakcrest elevation; however, individual variation should be evaluated
Cason & Lambert (1987) V: Supine backrest elevation at 0-20-40 degrees; Reference levels: phlebostatic axis or 10 cm from posterior chest at the angle of Louis: PA pressures Sample: N = 32 critically ill patients; n = 20 on MV without PEEP	Sequential backrest elevation at 0-20-40 degrees Two reference levels used at each backrest elevation End-expiration Digital data over a 10 second period, corrected for respiatory variation Stabilization period: 5 minutes	Clinically significant changes in PA pressures occurred in 13/32 patients at 20 degrees Clinically significant changes in PADP occurred in 17/32 patients at 40 degrees No significant differences were associated with the two reference levels Mean PADP significantly different at 40 degrees ($p < 0.04$)	Nonstandardized stabilization period at each backrest elevation Use of digital data No measurement of PAWP Did not specify AP diameter of the chest	Despite nonsignificant changes in PA pressures, large individual variations occurred; therefore, no change in practice can be recommended

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Cason, Lambert, Holland, & Huntsman (1990) V: Supine backrest elevation at 0 and 30 degrees; lateral position at 30 degrees; PA pressures Sample: N = 16 cardiac patients	Control: Flat, supine position Placed into right or left lateral position (30 degrees) Supine position with backrest elevation Placed into right or left lateral position (30 degrees lateral, 30 degree backrest elevation) Reference:Supine: 4th ICS/midaxillary line; Lateral: 4th ICS and midsternum Stabilization period: 5 minutes Individual fluctuation used as a control for clinical significance	PASP significantly affected by position (p = 0.0005) and elevation (p = 0.0001) No significant interaction between elevation and position (p = 0.83) PAMP significantly affected by position (p < 0.001) and elevation (p = 0.0194) PADP significantly affected by position (p < 0.001), but not backrest elevation __Change greater than individual fluctuation__ __0 degrees__ __PASP__ Right lateral 56.25% Left lateral 50.00% __PADP__ Right lateral 62.5% Left lateral 56.3% __30 degrees__ __PASP__ Right lateral 25.0% Left lateral 31.5% __PADP__ Right lateral 56.3% Left lateral 43.7%	Did not provide data regarding absolute changes in individual data No information with regard to method of data collection (analog vs digital) No repeated measures Did not control for sequential position change effect	Large variations occurred when compared with individual norm; although absolute values not specified Placement of the patient in the 30 degree lateral position with the backrest flat or at 30 degrees elevation is not recommended for PA pressure measurement

APPENDIX E

Effect of Lateral Position on

Pulmonary Artery Pressure Measurement

List of Abbreviations

ICS	intercostal space
L	left
MICU	medical intensive care unit
MV	mechanical ventilation
PA	pulmonary artery
PAD	pulmonary artery diastolic
PAM	pulmonary artery mean
PAS	pulmonary artery systolic
PAWP	pulmonary artery wedge pressure
R	right
RA	right atrial
RAP	right atrial pressure
S	supine
SICU	surgical intensive care unit
V	variables

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Murphy (1977) V: Supine, right and left lateral decubitus position; PA pressures Sample: $N = 8$ critically ill patients	Sequential position change: A1: S-L-S A2: L-S-L B1: S-R-S B2: R-S-R Total time: 15 minutes Repeated X 3 every 2 hours Pressures read at one and five minutes Analog data Reference: Supine: 4th ICS/ midanteroposterior diameter Lateral: 4th ICS/ midline	Mean differences PASP: $p > 0.05$ PADP $p > 0.05$ PAMP $p > 0.05$ PAWP $p\ 0.05$ 82% of PAWP differences < 4 torr All clinically significant changes in PAWP occurred in patients with control PAWP > 20 torr Correlation PADP-PAWP at one and five minutes $r = 0.87$, $p < 0.01$ Correlation PADP-PAWP S-R: $r = 0.94$, $p < 0.01$ S-L: $r = 0.90$, $p < 0.01$	Did not specify when readings were taken during the ventilatory cycle Pillows used to stabilize in the lateral position Small sample size PAWP > PADP in several cases, but did not specify the method for interpreting the PADP or PAWP	18% of patients demonstrated clinically significant changes; therefore, pressures should be measured in both positions and compared

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Whitman (1982) V: 20 degrees right and left lateral position, and supine position; PA pressures Sample: N = 50 cardiac surgery patients	Randomly assigned to one of six sequences of three positions Stabilization period: 15 minutes in each position Pressures repeated X 2 and averaged Total data collection time: 60-75 minutes Reference level: Supine: phlebostatic axis Lateral: not specified	Mean changes Supine to lateral PASP: $p > 0.05$ PADP: $p < 0.05$ PAMP: $p > 0.05$ RAP: $p < 0.05$ Individual differences (not provided in report) PASP: 0-20, mean 4.39 PADP: 0-11, mean 2.68 PAMP: 0-9, mean 2.3	Small sample for each data group Stabilized with pillows in lateral position Total collection time 60 to 75 minutes (could affect stability of the measurements) Did not specify method of data collection (graphic vs digital) Did not specify lateral reference point	Despite small mean changes, large individual changes occurred. Due to limitations of the study no change in practice is recommended
Bryant & Kennedy (1982) V: Supine, right and left lateral decubitus position; PA pressures Sample: N = 25 ICU patients	Randomized position changes from S-R-L lateral decubitus Reference: Supine: 4th ICS /midaxillary Lateral: 4th ICS /midsternum or mid-spinal column	Mean pressure differences PASP: +1 torr PADP: 0 torr PAMP: -1 torr PAWP: 0 torr Maximum change in PA and PAWP was 2-3 torr	Group data only Did not specify stabilizaiton period, method of stabilization, or method for reading the waveforms	Reported individual changes were small; therefore, reproducible PA pressure measurements may be obtained in the lateral position, with the transducer referenced to the 4th ICS/midsternum

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Wild (1983) V: Flat, supine, and 30 degree right and left lateral positions; PA pressures Sample: N = 30 critically ill patients	Sequential position change Group A: S-L-R-S Group B: S-R-L-S Reference: Supine: Phlebostatic axis Lateral: 4th ICS and midsternum Stabilization period: 3 minutes Analog data using one representative respiratory cycle Used soft wedge to stabilize in the lateral position	Range of mean pressures differences PASP: 3.6-3.8 ($p < 0.01$) PADP: 3.2-4.4 ($p < 0.01$) PAMP: 3.6-4.4 ($p < 0.01$) PAWP: 4.3-4.9 ($p < 0.01$) 28/30 patients had clinically significant changes 73% decrease in PAWP from supine-1 to either lateral position (mean 4.8 torr) 67% increase in PAWP from either lateral position to supine-2 (mean 4.0 torr) Supine vs lateral ($p < 0.01$) Group vs position ($p > 0.05$)	Correct identification of lateral position reference has not been determined	PA pressure measurements in the 30-degree lateral position is not recommended Further study to identify a reference level in the lateral position is needed

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Kennedy, Bryant, & Crawford (1984) V: Flat, supine and 90 degree right and left lateral position; PA pressures Sample: $N = 25$ critically ill patients	Randomized position change Used pillows to maintain lateral position Reference: Supine: 4th ICS midaxillary Lateral: 4th ICS midsternum or T4-T6 at midline of spinal column Analog data during one respiratory cycle; average between the peak and the trough Elapsed time between position change: 3 minutes	Maximum mean difference PASP: 0.3 ($p > 0.05$) PADP: 0.1 ($p > 0.05$) PAMP: -0.2 ($p > 0.05$) PAWP: 0.7 ($p > 0.05$) Individual variations All less than 2 torr	Used pillow to maintain position No repeated measures Used only medical patients Pressures not read at end-expiration	The sternum at the 4th ICS can be used to obtain reproducible measurements (compared to flat supine) The 90 degree lateral position can be used to obtain reproducible measurements

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Keating, Boylard, Eichler, & Reed (1986) V: Flat, supine and approximately 45 degrees right and left lateral position; PA pressures Sample: $N = 20$ critically ill patients (multisystem failure)	Sequential position change Supine-right-left-supine-right-left Stabilization period: 5 minutes Total time: 25 minutes Positioned at approximately 45 degrees with pillows Representative sample over one respiratory cycle Reference: Supine: Phlebostatic axis Lateral: 4th ICS/ midsternum Digital and analog data	Mean differences Right Left PASP: -6.7 -5.1 PADP: +7.4 -5.7 PAMP: -6.1 -6.4 All mean changes were statistically significant ($p < 0.0003$) Individual differences PASP: 3-17; mean 10.3 PADP: 3-20; mean 9.45 PAMP: 5-14; mean 9.84 No significant changes were noted between S-1 and S-2	Did not measure pressures at end-expiration Did not repeat flat, supine measurement at the completion of the study Position approximately 45 degrees Stabilization in lateral position with pillows	Lateral sidelying PA pressures were clinically and statistically different from flat, supine pressures Measurement of PA pressures in the 45 degree lateral position is not recommended The correct zero reference for lateral positioning needs to be determined

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Guenther, Kay, Cheng, & Lauer (1987) V: Flat, supine and right and left lateral decubitus positions; Flat, supine and prone position; PAWP and RAP Sample: *N* = 24; Group A *n* = 12 critically ill patients Group B: *n* = 12 laminectomy patients	Sequential position change in two groups of patients Group A: S-R-L Group B::S-prone Reference: Supine/prone: 4th ICS and midaxillary line Lateral: 4th ICS/ midsternum	**Mean pressure changes** Supine to Prone PAWP +6 RAP +1 Supine to Lateral PAWP +1 RAP -1 All pressure differences not statistically significant (authors note)	No data with respect to stabilization period, how data read (graphic vs digital), when read during the ventilatory cycle, how stabilized in the lateral position Group data only Small sample size Only measured PAWP and RAP	Without individual data a change in practice cannot be recommended Data that were identified as statistically nonsignificant exceeded expected fluctuation in the supine-prone group
Osika (1989) V: Flat, supine, and right and left lateral position with backrest elevation of 20,30, and 45 degrees; PA pressures Sample: *N* = 21 critically ill patient (81% on MV)	Each data set consisted of PA readings in the supine and right and left side-lying positions at one of three backrest elevations. PA pressure differences were analyzed for clinical significance Clinical significance: PASP/PADP/PAWP: > 5 torr PAWP: > 4 torr	**Clinically significant differences** 20 degrees: 71% 30 degrees: 57% 45 degrees: 78%	No data provided with rspect to reference level, method of stabilization, sequence effect, method of analyzing data (graphic vs digital), and length of time for data collection Did not specify degree of angle associated with sidelying position	Based on clinical changes, measurement of PA pressures in the "sidelying" position with backrest elevated cannot be recommended

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
Groos, Frisch, & Elliott (1990) V: Supine with backrest flat or 20 degrees, and lateral position with backrest flat or at 20 degrees; two reference levels; PA pressures Sample: _N_ = 59; _n_ = 30 SICU patients; _n_ = 29 MICU patients	Control: Whatever postion the patient was in at the beginning of study flat or 20-degrees supine or lateral) 73% SICU patients supine with backrest elevation of 20 degrees 79% MICU patients supine, flat Patients positioned at approximatley 45 degree lateral position, at whatever backrest postion they were found in Reference level: Supine: 4th ICS midaxillary line Lateral: SICU: 4th ICS dependent midaxillary MICU: 4th ICS midsternum Reference levels switched for MICU and SICU	Mean pressure differences PADP in supine-left statistically significant (_p_ < 0.01) in SICU group All other pressure changes nonsignificant All pressure changes in the MICU patients statistically significant (_p_ < 0.001) Individual pressure differences > 4 torr SICU: 7% MICU: 72% Lateral pressures were greater than supine when the transducer was at the 4th ICS/midsternum, and lower than supine at the 4th ICS/dependent midaxillary line Correlation supine to lateral position r/t transducer level #1 S-L: _r_ = 0.88, _p_ = 0.00 #2 S-L: _r_ = 0.77, _p_ < 0.01 #1 S-R: _r_ = 0.87, _p_ = 0.00 #2 S-R: _r_ = 0.64, _p_ < 0.05	Nonstandardized position Crossover data did not correct difference associated with the transducer position Positioned with pillows at approximately 45 degrees No interrater reliability No control of time between pressure measurements (every 2 hours or at the nurses' discretion) Method for data collection not specified (analog vs digital) Transducer held at various reference levels (not stabilized) Did not specify when pressures read during the ventilatory cycle	Due to large number of uncontrolled variables no recommendations can be made based on this study Determination of reference level in the lateral position needs to be determined

Investigator Variables/Sample	Methods	Findings	Limitations	Implications
	No special training for the nurses	**MICU**		
		#1 S-L: $r = 0.60$, $p = 0.001$		
		#2 S-L: $r = 0.66$, $p < 0.05$		
		#1 S-R: $r = 0.53$, $p = 0.88$		
		#2 S-R: $r = 0.71$, $p < 0.05$		

APPENDIX F

Clinical Simulation: Pulmonary Artery Pressure Measurement
Pilot Study Data

Clinical Simulation: Pulmonary Artery Pressure Measurement

Instructions: The questions that follow are related to events in the management of a patient with a myocardial infarction. The events are presented in the order that you would likely see in clinical practice. Therefore, please do not use information from later questions to assist in answering the earlier questions. To facilitate accurate assessment of your knowledge and ability to utilize information related to pulmonary artery pressure monitoring in a clinical scenario, do not use any references to assist you with completion of this test. Select the ONE best answer for each question.

Mark your answer on the mark sense answer sheet using a #2 pencil. DO NOT write your name on the mark sense sheet or the demographic data sheet. Return the answer sheet and the demographic data sheet in the enclosed envelope. Do not fold the mark sense sheet. Please keep the research questionnaire. If you would like an abstract of the research results and a copy of the correct answers please complete the attached form, and return it with the answer sheet and demographic data sheet. This information will be separated from your answer and demographic data sheets prior to scoring.

CLINICAL SCENARIO: Mr Jones is a 62-year old (75 kg, body surface area of 1.9 meters squared), who presents in the Emergency Room 12 hours after the onset of crushing substernal chest pressure. Mr Jones attributed the pressure to indigestion and treated the discomfort with antacids. On awakening this morning (0500), Mr Jones was diaphoretic, dizzy, and nauseated. His wife insisted that he come to the hospital. Admission vital signs include: arterial blood pressure 150/95 mm Hg (torr); heart rate 120 beats/minute; respiratory rate 30/minute and slightly labored. An electrocardiogram on admission demonstrated an anteroseptal wall myocardial infarction. In the Emergency Room, Mr Jones again experienced substernal chest pressure, refractory to sublingual nitroglycerin. Because Mr Jones had not urinated in 12 hours, and was unable to void, urinary bladder catheterization was performed, with a resultant drainage of 400 ml of urine. Mr Jones was transferred to the ICU/CCU, where the decision was made to place a pulmonary artery catheter to assist with therapeutic decision making.

SELECT ONE BEST ANSWER

1. The pulmonary artery (PA) catheter is placed to assist with

    therapeutic decision making and provides an indirect measurement

    of preload; PRELOAD reflects

    A. RESISTANCE TO EJECTION

    B. END-DIASTOLIC VOLUME

    C. END-SYSTOLIC VOLUME

    D. FORCE OF CONTRACTION

2. The PA catheter also provides indirect information related to

    vascular tone, which is reflected by:

    A. PRELOAD

    B. AFTERLOAD

    C. CONTRACTILITY

    D. EJECTION FRACTION

Questions 3 through 8 are based on the following data

Mr Jones's PA pressure readings were:

0700: PA - 32/23/27; PA wedge - 18; cardiac index - 2.4

0900: PA: 38/29/33; PA wedge - 23; cardiac index: 2.4

Between 0700 and 0900, Mr Jones developed bibasilar crackles,

and S3 and S4 heart sounds. At 0900, blood pressure was

100/60/73 torr; right atrial pressure was 11 torr; systemic

vascular resistance was 1088 dynes/sec/cm^{-5}, urine output 30

ml/hour.

3. The clinical findings at 0900 are consistent with

    A.. ORTHOSTATIC INTOLERANCE

    B. PRERENAL FAILURE

    C. PULMONARY EDEMA

    D. IMPAIRED SYSTEMIC PERFUSION

4. Your conclusion in question #3 is based on which of the

    following assumptions?

    A. THE RELATIONSHIP BETWEEN PERIPHERAL PERCUSION AND ORGAN

        FUNCTION

    B. THE RELATIONSHIP BETWEEN CARDIAC INDEX AND SYSTEMIC

        PERFUSION

    C. THE RELATIONSHIP BETWEEN ALTERED SYSTEMIC VASCULAR

        RESISTANCE AND THE INABILITY TO TOLERATE POSITION CHANGES

    D. THE RELATIONSHIP BETWEEN PA PRESSURES AND THE OCCURRENCE OF

        CLINICAL SIGNS OF PULMONARY CONGESTION

5. The ONE BEST clinical goal for Mr Jones based on the 0900

    hemodynamic values is to:

    A. INCREASE PRELOAD

    B. INCREASE CONTRACTILITY

    C. DECREASE AFTERLOAD

    D. DECREASE PRELOAD

6. In order to achieve the therapeutic goal in question #5, the

    most likely therapy would be

    A. VOLUME LOADING

    B. INOTROPIC THERAPY

    C. VOLUME REDUCTION

    D. CHRONOTROPIC THERAPY

7. At 0930 Mr Jones' hemodynamic values are: PA: 34/25/28;

    PAWP: 19; CI: 2.4 The changes between 0900 and 0930 may

    reflect all of the following EXCEPT:

    A. INCREASED CONTRACTILITY

    B. DECREASED AFTERLOAD

    C. INCREASED PRELOAD

    D. NORMAL FLUCTUATION

8. Based on the 0930 parameters, Mr Jones received a diuretic. At 1030 his PA pressures were: PA: 26/16/19; PAW: 12; CI: 2.3. He continues to have S3 and S4 heart sounds.

   You have a standing order: "Give Furosemide 10 mg IV push q 1 hour, to relieve pulmonary congestion." Based on the data you decide to:

   A. GIVE THE FUROSEMIDE, BECAUSE MR JONES STILL HAS CLINICAL SIGNS OF PULMONARY CONGESTION

   B. HOLD THE FUROSEMIDE BECAUSE PA PRESSURE CHANGES PROVIDE A MORE SENSITIVE AND TIMELY INDICATOR OF PULMONARY STATUS

   C. GIVE THE FUROSEMIDE, BECAUSE THE PA PRESSURES DO NOT ACCURATELY REFLECT THE PATIENTS CLINICAL STATUS

   D. HOLD THE FUROSEMIDE BECAUSE THE PA PRESSURE CHANGES ARE INDICATIVE OF HYPOVOLEMIA

9. You reposition Mr Jones before attaining his next set of hemodynamic readings. In order to ensure accurate readings, you reference the PA catheter system to the phlebostatic axis, which is defined as the:

   A. INTERSECTION OF AN AXIS TRANSECTING THE FOURTH INTERCOSTAL SPACE AT THE STERNUM AND THE MIDAXILLARY LINE

   B. POINT 5 CM BELOW THE STERNAL NOTCH

   C. INTERSECTION OF AN AXIS TRANSECTING THE FOURTH INTERCOSTAL SPACE AT THE STERNUM AND 1/2 THE DISTANCE BETWEEN THE ANTERIOR AND POSTERIOR SURFACE OF THE CHEST

   D. FOURTH INTERCOSTAL SPACE AT THE LEFT PARASTERNAL BORDER

Questions 10 through 12 are related to the following information

On his second day post-MI, Mr Jones extended his infarct laterally,

and subsequently demonstrated progressive cardiopulmonary

instability. Mr Jones was intubated and placed on a ventilator

(Settings: Oxygen (FiO2) - 0.60; Intermittent Mandatory Ventilation

Rate (IMV) - 10/minute; Tidal volume - 900 ml; Positive End-

Expiratory Pressure (PEEP) - 0 cm H_2O). The following set of

hemodynamic parameters were taken after initiation of mechanical

ventilation.
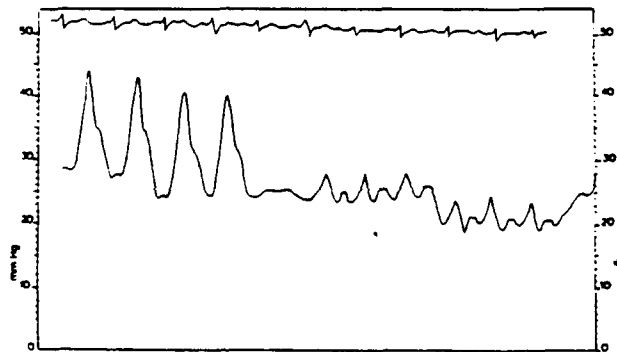
Additional hemodynamic data: arterial blood pressure: 88/48/61

torr; heart rate: 130 beats/minute; cardiac index: 1.5

liters/min/M^2; systemic vascular resistance: 1410 dynes/sec^{-5}; right

atrial pressure = 12 torr.

10. Based on the above pressure tracing, current pulmonary artery

    pressures are:

    A. PAS: 48; PAD: 26; PAM: 34; PAW: 24

    B. PAS: 44; PAD: 25; PAM: 33; PAW: 28

    C. PAS: 41; PAD: 25; PAM: 32; PAW: 22

    D. PAS: 41; PAD: 33; PAM: 35; PAW: 28

11. The hemodynamic parameters in question #10 are consistent

with the diagnosis of:

A. ACUTE PERICARDIAL TAMPONADE

B. MITRAL INSUFFICIENCY

C. PULMONARY EMBOLUS

D. LEFT VENTRICULAR FAILURE

12. Your evaluation of the characteristics of the "a" wave on

the pressure waveform include:

A. CHANGES INDICATIVE OF DECREASED MYOCARDIAL COMPLIANCE

B. CHANGES INDICATIVE OF MITRAL VALVE INSUFFICIENCY

C. CHANGES INDICATIVE OF INCREASED PULMONARY COMPLIANCE

D. CHANGES INDICATIVE OF TRICUSPID STENOSIS

13. Clinical findings associated with the hemodynamic parameters

may include:

A. NO PULMONARY CONGESTION; NO IMPAIRMENT IN PERIPHERAL

PERFUSION

B. PULMONARY CONGESTION; NO IMPAIRMENT IN PERIPHERAL PERFUSION

C. NO PULMONARY CONGESTION; IMPAIRMENT IN PERIPHERAL PERFUSION

D. PULMONARY CONGESTION; IMPAIRMENT IN PERIPHERAL PERFUSION

14. Therapeutic options, based on the information in #10 include

A. VOLUME AUGMENTATION

B. VASOCONSTRICTION

C. VOLUME REDUCTION

D. CHRONOTROPIC THERAPY

15. The goal of the therapy in #14 is a (an)

    A. INCREASE IN AFTERLOAD

    B. INCREASE IN HEART RATE

    C. INCREASE IN CONTRACTILITY

    D. DECREASE IN PRELOAD

16. Additional therapeutic options for Mr Jones include the use of

    all of the following EXCEPT

    A. MECHANICAL ASSIST DEVICE

    B. VASODILATOR THERAPY

    C. INOTROPIC THERAPY

    D. PRELOAD AUGMENTATION

17. In order to aid therapeutic decision making, you decide to

    construct a ventricular function curve, which compares left

    ventricular end-diastolic volume with cardiac work. Indices of

    left-ventricular end-diastolic volume include all of the

    following EXCEPT

    A. LEFT ATRIAL PRESSURE

    B. LEFT VENTRICULAR END-DIASTOLIC PRESSURE

    C. PULMONARY ARTERY WEDGE PRESSURE

    D. CENTRAL VENOUS PRESSURE

18. Indices of cardiac work include all of the following EXCEPT:

    A. STROKE VOLUME

    B. STROKE WORK

    C. EJECTION FRACTION

    D. CARDIAC INDEX

19. The optimal hemodynamic effects of the therapy outlined in question #14 would be reflected by movement from point X to point ___ on the ventricular function curve

A. A

B. B

C. C

D. D
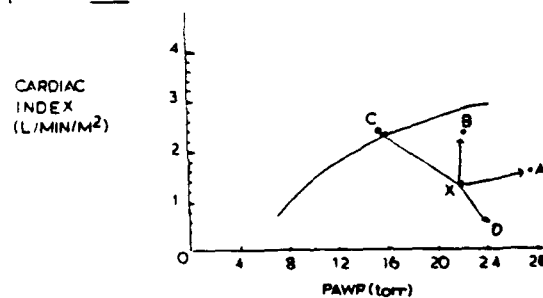
During continuous PA tracing monitoring you note the following waveform:
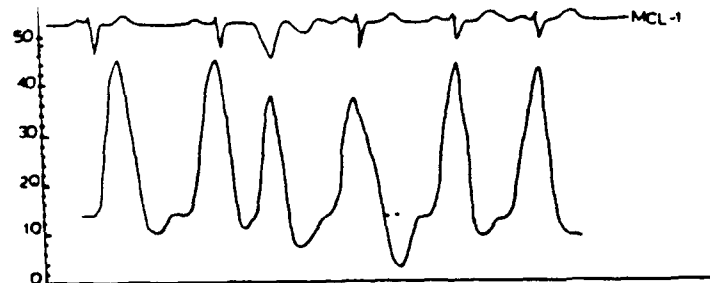
20. These data reflect

A. PULMONARY HYPERTENSION

B. LEFT VENTRICULAR FLUID OVERLOAD

C. PERICARDIAL TAMPONADE

D. RIGHT VENTRICULAR CATHETER PLACEMENT

21. Based on your assessment (#20) appropriate nursing actions would include:

    A. ASSESSMENT OF THE DYNAMIC RESPONSE

    B. ADMINISTRATION OF PRN DIURETIC

    C. INFLATION OF PA CATHETER BALLOON

    D. ASSESSMENT FOR PULSUS PARADOXUS

Following resolution of the problem described in #20-21, you decide to evaluate the dynamic-response characteristics of the PA catheter system. The response to a stepped pressure change is:
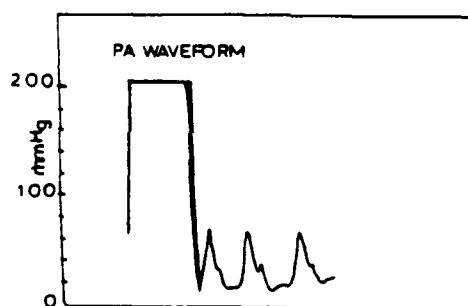
22. Actions required based on this tracing include

    A. CHECKING THE SYSTEM FOR AIR BUBBLES AND KINKS

    B. ADDING ADDITIONAL TUBING, OR A DAMPING DEVICE

    C. RECALIBRATING AND REZEROING THE PA CATHETER SYSTEM

    D. CONTINUING TO PERIODICALLY EVALUATE THE SYSTEM

23. Despite mechanical ventilation, and the administration of 100%
    oxygen, Mr Jones continues to have poor tissue oxygenation;
    therefore, 10 cm H2O of PEEP is progressively added. The
    addition of PEEP will

    A. INVALIDATE THE RELATIONSHIP BETWEEN PAW AND LA PRESSURES

    B. NOT EFFECT THE RELATIONSHIP BETWEEN PAW AND LA PRESSURES

    C. HAVE AN EFFECT ON THE PAW AND LA PRESSURE RELATIONSHIP, IF
       THE PATIENT HAS DECREASED LUNG COMPLIANCE

    D. HAVE AN EFFECT ON THE PAW AND LA PRESSURE RELATIONSHIP, IF
       THE PA CATHETER IS PLACED BELOW THE LA

24. Mr Jones has finally fallen asleep. He is currently positioned
    on his right side (approximately 30 degrees from supine), with
    the head of the bed elevated 20 degrees. A set of hemodynamic
    pressure readings were taken. PA: 40/32/34; PAW: 30. You
    conclude these parameters are

    A. REFLECTIVE OF A FURTHER DECREASE IN CARDIAC FUNCTION

    B. REFLECTIVE OF PULMONARY DETERIORATION

    C. INACCURATE DUE TO THE PATIENT'S POSITION

    D. ACCURATE BECAUSE THE TRANSDUCER WAS REFERENCED CORRECTLY

25. Select the reason you would use to support your conclusion in #24.

   A. AN INCREASE IN PAW PRESSURE MAY BE REFLECT A FURTHER DETERIORATING CARDIAC FUNCTION

   B. THE ELEVATION OF THE PULMONARY ARTERY SYSTOLIC AND DIASTOLIC PRESSURE IS REFLECTIVE OF PULMONARY DYSFUNCTION

   C. PULMONARY ARTERY PRESSURE MEASUREMENTS ARE NOT CONSISTENTLY REPRODUCIBLE IN THE SIDE-LYING POSITION

   D. PA PRESSURE MEASUREMENTS ARE CONSISTENTLY REPRODUCIBLE IN ALL SIDE-LYING POSITIONS, AS LONG AS THE CATHETER IS CORRECTLY REFERENCED STERNUM

During continuous pulmonary artery pressure monitoring you note the following pressure tracing:
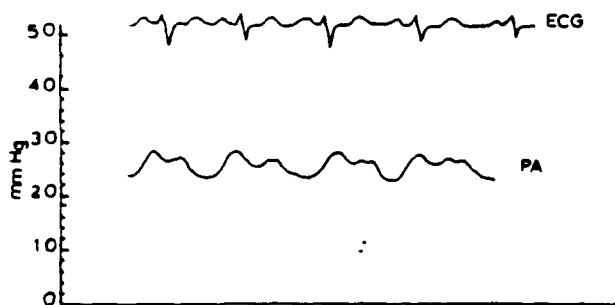
26. This pulmonary artery pressure tracing is indicative of:

   A. LEFT VENTRICULAR FAILURE

   B. SPONTANEOUS WEDGING

   C. A DECREASE IN PRELOAD

   D. ACUTE MITRAL INSUFFICIENCY

27. Required nursing actions based on your analysis in #23 include

   A. FLUSHING THE SYSTEM WITH THE FAST-FLUSH DEVICE

   B. HAVING MR JONES TURN ONTO HIS SIDE OR MOVE HIS ARM

   C. WEANING THE VASODILATOR THERAPY

   D. INFLATING THE BALLOON TO EVALUATE THE PAW PRESSURE

28. Your current nursing plan of care for Mr Jones, with regard to

   PA pressure monitoring includes:

   A. PLACING HIM FLAT AND SUPINE FOR PA PRESSURE MEASUREMENTS

   B. REMOVING HIM FROM THE VENTILATOR FOR PA PRESSURE

      MEASUREMENTS

   C. COMPARING FLAT, SUPINE PA PRESSURE MEASUREMENTS WITH SUPINE,

      BACKREST UPRIGHT PRESSURE MEASUREMENTS FOR CONSISTENCY

   D. AVERAGING PA PRESSURE MEASUREMENTS OVER SEVERAL RESPIRATORY

      CYCLES

Thank you for your assistance with this project. Please return the

answer sheet and demographic data sheet in the envelope provided.

If you would like a copy of the correct answers, rationale, and an

abstract of the results of this study, please include your name and

address on the form at the bottom of this page and include it in the

return envelope. To ensure anonymity, this personal information

will be separated from the answer sheet and demographic sheet prior

to scoring

LENGTH OF TIME TO COMPLETE EXAMINATION _____(MINUTES)

CONTENT AREAS YOU⁴LIKE ADDED TO THE EXAMINATION

QUESTIONS YOU THINK SHOULD BE DELETED FROM THE EXAMINATION (Please

include rationale)

QUESTIONS YOU HAD DIFFICULTY ANSWERING (Please include rationale for

difficulty)

Thank you again for your assistance, it is GREATLY appreciated.

Table F-1.

Answer Key - Pilot Study

1.	B	24.	C
2.	B	25.	C
3.	C	26.	C
4.	D	27.	B
5.	D	28.	C
6.	C		
7.	C		
8.	B		
9.	C		
10.	C		
11.	D		
12.	A		
13.	D		
14.	C		
15.	D		
16.	D		
17.	D		
18.	C		
19.	C		
20.	D		
21.	C		
22.	D		
23.	B		

Table F-2.

Test Scores - Frequencies and Distributions

Item Number	Weight	Means	Frequencies		Distribution (# equals 2.5%)
1	A. ----	----	0	0.0%	
	B. 1.00	14.50	18	90.0%	###################################
	C. ----	17.00	2	10.0%	####
	D. ----	----	0	0.0%	
2	A. ----	-----	-0	0.0%	
	B. 1.00	16.15	13	65.0%	##########################
	C. ----	13.00	3	15.0%	######
	D. ----	12.75	4	20.0%	########
3	A. ----	-----	0	0.0%	
	B. ----	-----	0	0.0%	
	C. 1.00	14.95	19	95.0%	######################################
	D. ----	10.00	1	5.0%	##
4.	A. ----	10.00	1	5.0%	##
	B. ----	-----	0	0.0%	
	C. ----	-----	0	0.0%	
	D. 1.00	14.95	19	95.0%	######################################
5.	A. ----	-----	0	0.0%	
	B. ----	17.75	4	20.0%	########
	C. ----	13.00	7	35.0%	##############
	D. 1.00	15.78	9	45.0%	##################
6.	A. ----	12.00	1	5.0%	##
	B. ----	16.25	4	20.0%	########
	C. 1.00	14.54	13	65.0%	##########################
	D. ----	16.00	1	5.0%	##
7.	A. ----	14.00	3	15.0%	######
	B. ----	16.00	1	5.0%	##
	C. 1.00	16.78	9	45.0%	##################
	D. ----	12.67	6	30.0%	############
8.	A. ----	17.80	5	25.0%	##########
	B. 1.00	13.75	8	40.0%	################
	C. ----	-----	0	0.0%	
	D. ----	14.67	6	30.0%	############
9.	A. ----	14.83	6	30.0%	############
	B. ----	-----	0	0.0%	
	C. 1.00	15.00	14	70.0%	############################
	D. ----	-----	0	0.0%	

Item Number	Weight	Means	Frequencies		Distribution (# equals 2.5%)
10.	A. ----	10.50	2	10.0%	####
	B. ----	14.20	5	25/0%	·#########
	C. 1.00	17.20	10	50.0%	####################
	D. ----	16.00	1	5.0%	##
11.	A. ----	14.00	3	15.0%	######
	B. ----	11.00	1	5.0%	##
	C. ----	-----	0	0.0%	
	D. 1.00	15.25	16	80.0%	################################
12.	A. 1.00	13.91	11	55.0%	######################
	B. ----	17.60	5	25.0%	##########
	C. ----	17.33	3	15.0%	######
	D. ----	-----	0	0.0%	
13.	A. ----	9.00	1	5.0%	##
	B. ----	21.00	1	5.0%	##
	C. ----	17..00	2	10.0%	####
	D. 1.00	14.56	16	80.0%	################################
14.	A. ----	13.25	4	20.0%	########
	B. ----	10.00	1	5.0%	##
	C. 1.00	16.71	7	35.0%	##############
	D. ----	16.71	7	35.0%	##############
15.	A. ----	10.50	2	10.0%	####
	B. ----	-----	0	0.0%	
	C. ----	15.56	9	45.0%	##################
	D. 1.00	15.89	9	45.0%	##################
16.	A. ----	-----	0	0.0%	
	B. ----	13.50	6	30.0%	############
	C. ----	15.50	2	10.0%	####
	D. 1.00	15.75	12	60.0%	########################
17.	A. ----	14.67	3	15.0%	######
	B. ----	14.00	2	10.0%	####
	C. ----	15.00	4	20.0%	########
	D. 1.00	15.45	11	55.0%	######################
18.	A. ----	19.00	1	5.0%	##
	B. ----	14.29	14	70.0%	############################
	C. 1.00	19.00	2	10.0%	####
	D. ----	18.00	3	15.0%	######

Item Number	Weight	Means	Frequencies		Distribution (# equals 2.5%)
19.	A. ----	14.00	1	5.0%	##
	B. ----	11.67	3	15.0%	######
	C. 1.00	16.00	15	75.0%	##############################
	D. ----	-----	0	0.0%	
20.	A. ----	15.00	4	20.0%	########
	B. ----	12.50	4	20.0%	########
	C. ----	18.50	2	10.0%	####
	D. 1.00	16.44	0	45.0%	##################
21.	A. ----	-----	0	0.0%	
	B. ----	13.60	5	25.0%	##########
	C. 1.00	16.30	10	50.0%	####################
	D. ----	14.40	5	25.0%	##########
22.	A. ----	12.00	3	15.0%	######
	B. ----	-----	0	0.0%	
	C. ----	16.45	11	55.0%	######################
	D. 1.00	16.40	5	25.0%	##########
23.	A. ----	19.00	3	15.0%	######
	B. 1.00	17.00	3	15.0%	######
	C. ----	14.87	8	40.0%	################
	D. ----	14.80	5	25.0%	##########
24.	A. ----	14.00	3	15.0%	######
	B. ----	12.50	2	10.0%	####
	C. 1.00	16.20	10	50.0%	####################
	D. ----	12.00	3	15.0%	######
25.	A. ----	14.00	3	15.0%	######
	B. ----	12.50	2	10.0%	####
	C. 1.00	16.20	10	50.0%	####################
	D. ----	12.00	3	15.0%	######
26.	A. ----	12.00	1	5.0%	##
	B. 1.00	16.20	15	75.0%	##############################
	C. ----	11.50	2	10.0%	####
	D. ----	11.00	1	5.0%	##
27.	A. ----	16.33	3	15.0%	######
	B. 1.00	16.90	10	50.0%	####################
	C. ----	10.50	2	10.0%	####
	D. ----	13.75	4	20.0%	########

28.   A. ---- 15.40   5 25.0%   ##########
             B. ---- -----   0   0.0%
             C. 1.00 15.80 10 50.0%   ####################
             D. ---- 14.75   4 20.0%   #########

Table F-3.

Group Data - Total and Subset Scores

Score Name	Total Score	Average Score	Standard Deviation	Largest Score	Smallest Score
Total Test	28	15.65	3.82	21.00	9.00
Clinical	17	10.35	2.08	14.00	5.00
Technical	7	3.10	1.73	6.00	0.00
Complications	4	2.20	1.44	4.00	0.00
Cognitive Level I	7	4.15	1.46	7.00	1.00
Cognitive Level II	12	7.15	1.90	10.00	3.00
Cognitive Level III	9	4.35	1.31	7.00	2.00

Table F-4.

Score Correlations

	Total	Clinical	Technical	Complications	Level I	Level II	Level III
Total	1.00	0.79	0.67	0.71	0.79	0.92	0.70
Clinical	0.79	1.00	0.21	0.40	0.46	0.79	0.63
Technical	0.67	0.21	1.00	0.27	0.83	0.56	0.23
Complications	0.71	0.40	0.27	1.00	0.44	0.63	0.68
Level I	0.79	0.46	0.83	0.44	1.00	0.64	0.26
Level II	0.92	0.79	0.56	0.63	0.64	1.00	0.52
Level III	0.70	0.63	0.23	0.68	0.26	0.52	1.00

Table F-5.

Test Reliability: Master versus Nonmaster

		First Test		
		Master	Nonmaster	Total
	Master	2 (0.22)	2 (0.22)	4 (0.44)
Second Test	Nonmaster	0 (0.00)	5 (0.55)	5 (0.55)
	Total	2 (0.22)	7 (0.78)	9 (1.00)

$Po = 0.77$

$P_c = 0.52$

Kappa = 0.52

$Kappa_{max} = 0.52$

$Kappa/Kappa_{max} = 1.00$

Table F-6.

Item Difficulty/Discrimination

Item on	Critical Care Nurses	Undergraduate/ Novice Nurses	Item Difficulty Contrasted Group Difficulty Index	Statistical Significance	Total Item Difficulty	Item Discriminati
1	0.90	0.91	-0.01	NS	90/Easy	-0.19/Poor
2	0.90	0.54	0.36	p < 0.05	65/Medium	0.44/Good
3	1.00	0.91	0.09	NS	95/Easy	0.29/Fair
4	1.00	0.91	0.09	NS	95/Easy	0.29/Fair
5	0.60	0.36	0.24	NS	45/Hard	0.14/Fair
6	0.70	0.63	0.07	NS	65/Medium	-0.16/Poor
7	0.70	0.27	0.43	NS	45/hard	0.40/Good
8	0.50	0.54	-0.04	NS	40/Hard	-0.31/Poor
9	0.80	0.64	0.16	NS	70/Medium	0.02/Poor
10	0.80	0.36	0.44	p < 0.05	50/Hard	0.58/Good
11	1.00	0.54	0.46	p < 0.05	80/Medium	0.22/Fair
12	0.30	0.73	-0.43	NS	55/Medium	-0.33/Poor
13	0.80	0.73	0.07	NS	80/Medium	-0.15/Poor
14	0.50	0.27	0.23	NS	35/Hard	0.28/Fair
15	0.50	0.45	0.05	NS	45/Hard	0.17/Fair
16	0.80	0.36	0.44	NS	60/Medium	0.23/Fair
17	0.70	0.36	0.34	NS	55/Medium	0.10/Fair
18	0.20	0.00	0.20	NS	10/Hard	0.31/Good
19	1.00	0.54	0.46	p < 0.05	75/Medium	0.53/Good
20	0.80	0.18	0.12	p < 0.05	45/Hard	0.31/Good
21	0.80	0.27	0.53	p < 0.05	50/Hard	0.32/Good
22	0.30	0.18	0.12	NS	25/Hard	0.15/Fair
23	0.20	0.00	0.20	NS	15/Hard	0.17/Fair
24	0.70	0.36	0.34	NS	50/Hard	0.29/Fair
25	0.70	0.36	0.34	NS	50/Hard	0.29/Fair
26	1.00	0.54	0.46	p < 0.05	75/Medium	0.64/Good
27	0.70	0.36	0.34	NS	50/Hard	0.49/Good
28	0.60	0.36	0.34	NS	50/Hard	0.18/Fair

Table F-7.

Item-Objective Congruence

Item	Congruence Value	Content Area	Congruence Value	Cognitive Level
1	1.00	Clinical	1.00	Level I
2	1.00	Clinical	1.00	Level I
3	1.00	Clinical	0.50	Level II
4.	1.00	Clinical	0.42	Level II
5.	1.00	Clinical	0.50	Level III
6.	1.00	Clinical	0.17	Level III
7.	1.00	Clinical	0.50	Level III
8.	0.75	Clinical	0.42	Level III
9.	1.00	Technical	1.00	Level I
10.	0.25	Technical	0.00	Level II
11.	0.88	Clinical	0.50	Level II
12.	1.00	Clinical	0.00	Level III
13	1.00	Clinical	0.00	Level II
14.	1.00	Clinical	0.92	Level III
15.	1.00	Clinical	0.50	Level II
16.	1.00	Clinical	0.42	Level II
17.	1.00	Clinical	0.50	Level I
18.	1.00	Clinical	0.50	Level I
19.	1.00	Clinical	0.50	Level II
20.	0.00	Complication	0.00	Level II
21.	0.00	Complications	0.42	Level II
22.	1.00	Technical	0.50	Level II
23.	0.00	Technical	0.50	Level II
24.	0.50	Technical	1.00	Level III
25.	0.50	Technical	0.50	Level II
26.	0.00	Complications	0.50	Level I
27.	0.00	Complications	0.92	Level II
28.	1.0	Technical	0.92	Level II

Average Congruency Percentage

Cognitive Levels	Content Area
Rater A = 0.82	Rater A = 1.0
Rater B = 0.68	Rater B = 0.71
Rater C = 0.61	
Average = 0.70	Average = 0.86

Table F-8.

Blueprint - Pilot Study

	LEVEL I KNOWLEDGE/ COMPREHENSION	LEVEL II APPLICATION/ ANALYSIS	LEVEL III SYNTHESIS/ EVALUATION	TOTAL
CLINICAL	1,2,17,18	3,4,11,13, 15,16,19	5,6,7,8, 12,14	17 (61%)
TECHNICAL	9,10	22,23,25	24,28	7 (25%)
COMPLICATIONS	26	20,27	21	4 (14%)
TOTAL	7(25%)	12 (43%)	9 (32%)	28 (100%)

Table F-9.

<u>Scoring - Pilot Study Subject</u>

	LEVEL I KNOWLEDGE/ COMPREHENSION	LEVEL II APPLICATION/ ANALYSIS	LEVEL III SYNTHESIS/ EVALUATION	TOTAL
CLINICAL	1,2,17,18	3,4,11,13, 15,16,19	⑤,⑥ 7,⑧ 12,14	14/17 (82%)
TECHNICAL	9,10	㉒,㉓ 25	24,28	5/7 (71%)
COMPLICATIONS	26	⑳,27	㉑	2/4 (50%)
TOTAL	7/7(100%)	9/12(75%)	5/9(56%)	28 (100%)

Note. ◯= INCORRECT

Table F-10.

Content Validity - Item Relevance

		Judge 1		
		Not/Somewhat Relevant	Quite/Very Relevant	Total
Judge 2	Not/Somewhat Relevant	25 (0.30)	1 (0.01)	26 (0.31)
	Quite/Very Relevant	33 (0.39)	25 (0.30)	58 (0.69)
	Total	58 (0.69)	26 (0.31)	84 (1.00)

P_O = 0.60

P_C = 0.42

Kappa = 0.31

$Kappa_{max}$ = 0.33

$Kappa/Kappa_{max}$ = 0.94

APPENDIX G

Clinical Simulation: Pulmonary Artery Pressure Measurement

Clinical Simulation: Pulmonary Artery Pressure Measurement

Instructions: The questions that follow are related to events in the management of a patient with a myocardial infarction. The events are presented in the order that you would likely see in clinical practice. Therefore, please do not use information from later questions to assist in answering the earlier questions. To facilitate accurate assessment of your knowledge and ability to utilize information related to pulmonary artery pressure monitoring in a clinical scenario, do not use any references to assist you with completion of this test. Select the ONE best answer for each question.

Mark your answer on the mark sense answer sheet using a #2 pencil. DO NOT write your name on the mark sense sheet or the demographic data sheet. Return the answer sheet and the demographic data sheet in the enclosed envelope. Do not fold the mark sense sheet. Please keep the research questionnaire. If you would like an abstract of the research results and a copy of the correct answers please complete the attached form, and return it with the answer sheet and demographic data sheet. This information will be separated from your answer and demographic data sheets prior to scoring.

:

:

CLIP

....

NAME (please print)

ADDRESS

CLIP

7. At 0930 Mr Jones' hemodynamic values were: PA: 34/25; PA mean: 26; PA wedge: 19; CI: 2.4 L/min/M^2. The changes between 0900 and 0930 may reflect all of the following EXCEPT:
   A. INCREASED CONTRACTILITY
   B. DECREASED AFTERLOAD
   C. INCREASED PRELOAD
   D. NORMAL FLUCTUATION

8. Based on the 0930 parameters, Mr Jones received a diuretic. At 1030 his PA pressures were: PA: 26/16; PA mean: 19; PA wedge: 12; CI: 2.3 L/min/M^2. He continued to have S3 and S4 heart sounds. You have a standing order: "Give Furosemide 10 mg IV push q 1 hour, to relieve pulmonary congestion." Based on the data you decide to:
   A. GIVE THE FUROSEMIDE, BECAUSE MR JONES STILL HAS CLINICAL SIGNS OF PULMONARY CONGESTION
   B. HOLD THE FUROSEMIDE BECAUSE PA PRESSURE CHANGES PROVIDE A MORE SENSITIVE AND TIMELY INDICATOR OF PULMONARY STATUS
   C. GIVE THE FUROSEMIDE, BECAUSE THE PA PRESSURES DO NOT ACCURATELY REFLECT THE PATIENTS CLINICAL STATUS
   D. HOLD THE FUROSEMIDE BECAUSE THE PA PRESSURE CHANGES ARE INDICATIVE OF SEVERE HYPOVOLEMIA

9. You reposition Mr Jones before attaining his next set of hemodynamic readings. In order to ensure accurate readings, you reference the PA catheter system to the phlebostatic axis, which is defined as the:
   A. INTERSECTION OF AN AXIS TRANSSECTING THE FOURTH INTERCOSTAL SPACE AT THE STERNUM AND THE MIDAXILLARY LINE
   B. POINT 5 CM BELOW THE STERNAL NOTCH
   C. INTERSECTION OF AN AXIS TRANSSECTING THE FOURTH INTERCOSTAL SPACE AT THE STERNUM AND 1/2 THE DISTANCE BETWEEN THE ANTERIOR AND POSTERIOR SURFACE OF THE CHEST
   D. FOURTH INTERCOSTAL SPACE AT THE LEFT PARASTERNAL BORDER

During continuous pulmonary artery pressure monitoring you note the following pressure tracing:
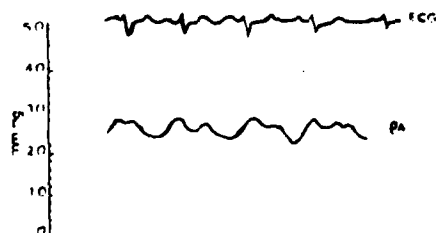
10. This pulmonary artery pressure tracing is indicative of: _
    A. PERICARDIAL TAMPONADE
    B. SPONTANEOUS WEDGING
    C. A DECREASE IN PRELOAD
    D. ACUTE MITRAL INSUFFICIENCY

11. Required nursing actions based on your analysis in #10 include:
    A. VIGOROUSLY FLUSHING THE SYSTEM WITH THE FAST-FLUSH DEVICE
    B. HAVING MR JONES TURN ONTO HIS SIDE OR MOVE HIS ARM
    C. WEANING THE VASODILATOR THERAPY
    D. INFLATING THE BALLOON TO EVALUATE THE PA WEDGE PRESSURE

Questions 12 through 20 are related to the following information:

On his second day post-MI, Mr Jones extended his infarct laterally, and subsequently demonstrated progressive cardiopulmonary instability. Mr Jones was intubated and placed on a ventilator (Settings: Oxygen (FIO2) - 0.60; Intermittent Mandatory Ventilation Rate (IMV)-10/minute; Tidal volume - 900 ml; Positive End-Expiratory Pressure (PEEP) - 0 cm H_2O). The following set of hemodynamic parameters were taken after initiation of mechanical ventilation.
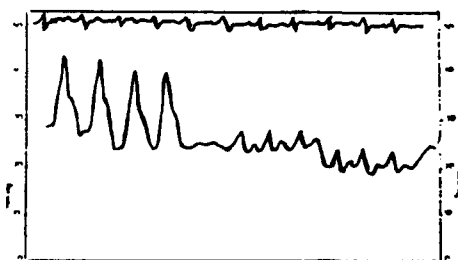
Additional hemodynamic data: arterial blood pressure: 86/48 torr; mean arterial pressure: 61 torr; heart rate: 130 beats/minute; cardiac index: 1.8 L/min/M^2; systemic vascular resistance: 1410 dynes/sec^{-5}; right atrial pressure = 12 torr.

12 Based on the above pressure tracing, the pulmonary artery SYSTOLIC pressure is:

A. 44

B. 41

C. 32

D. 28

13 Based on the above pressure tracing, the pulmonary artery DIASTOLIC pressure is:

A. 19

B. 23

C. 25

D. 28

14 Based on the above pressure tracing, the pulmonary artery MEAN pressure is:

A. 35

B. 32

C. 30

D. 26

15 Based on the above pressure tracing, the pulmonary artery WEDGE pressure is:

A. 2

B. 25

C. 22

D. 19

16 The hemodynamic parameters in the above tracing are consistent with the diagnosis of:

A. ACUTE PERICARDIAL TAMPONADE

B. MITRAL INSUFFICIENCY

C. PULMONARY EMBOLUS

D. LEFT VENTRICULAR FAILURE

17 Clinical findings associated with the above hemodynamic parameters may include:

A. NO PULMONARY CONGESTION; NO IMPAIRMENT IN SYSTEMIC PERFUSION

B. PULMONARY CONGESTION; NO IMPAIRMENT IN SYSTEMIC PERFUSION

C. NO PULMONARY CONGESTION; IMPAIRMENT IN SYSTEMIC PERFUSION

D. PULMONARY CONGESTION; IMPAIRMENT IN SYSTEMIC PERFUSION

(TURN PAGE OVER)

18. Therapeutic options, based on the information in the clinical scenario and hemodynamic parameters (data from questions #12-15) include :

A. VOLUME AUGMENTATION
B. VASOCONSTRICTION
C. VOLUME REDUCTION
D. CHRONOTROPIC THERAPY

19. The goal of the therapy in #18 is a (an):

A. INCREASE IN AFTERLOAD
B. INCREASE IN HEART RATE
C. INCREASE IN CONTRACTILITY
D. DECREASE IN PRELOAD

20. Additional therapeutic options for Mr Jones include the use of all of the following EXCEPT:

A. MECHANICAL ASSIST DEVICE
B. VASODILATOR THERAPY
C. INOTROPIC THERAPY
D. PRELOAD AUGMENTATION

21. In order to aid therapeutic decision making, you decide to construct a ventricular function curve, which compares left ventricular end-diastolic volume with cardiac work. Indices of left-ventricular end-diastolic volume include all of the following EXCEPT:

A. LEFT ATRIAL PRESSURE
B. LEFT VENTRICULAR END-DIASTOLIC PRESSURE
C. PULMONARY ARTERY WEDGE PRESSURE
D. CENTRAL VENOUS PRESSURE

22. Indices of cardiac work include all of the following EXCEPT:

A. STROKE VOLUME
B. STROKE WORK
C. PULMONARY ARTERY WEDGE PRESSURE
D. CARDIAC INDEX

23. The optimal hemodynamic effects of the therapy outlined in questions #18-20 would be reflected by movement from point X to point ___ on the ventricular function curve:

A. A
B. B
C. C
D. D
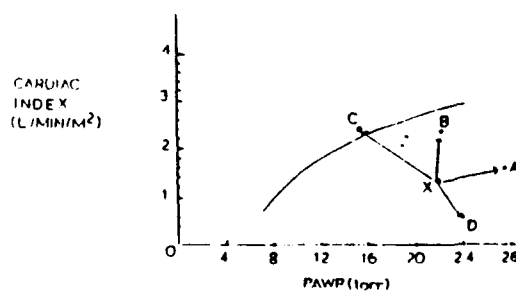
During continuous PA tracing monitoring you note the following waveform:

24. These data reflect:
    A. PULMONARY HYPERTENSION
    B. LEFT VENTRICULAR FLUID OVERLOAD
    C. PERICARDIAL TAMPONADE
    D. RIGHT VENTRICULAR CATHETER PLACEMENT

25. Based on your assessment (#24) appropriate nursing actions would include:
    A. ASSESSMENT OF THE DYNAMIC RESPONSE
    B. ADMINISTRATION OF PRN DIURETIC
    C. INFLATION OF PA CATHETER BALLOON
    D. ASSESSMENT FOR PULSUS PARADOXUS

26. Despite mechanical ventilation, and the administration of 100% oxygen, Mr Jones continues to have poor tissue oxygenation; therefore, 10 cm H2O of PEEP is progressi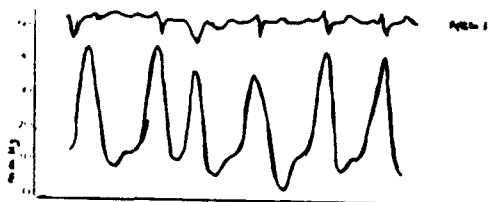vely added. Current PA pressure readings are: PA: 40/32; PA mean: 34; PA wedge: 30. Based on this information, the true PA wedge pressure is APPROXIMATELY:
    A. 34
    B. 30
    C. 26
    D. 20

27. Mr Jones has finally fallen asleep. He is currently positioned on his right side (approximately 30 degrees from flat, supine), with the head of the bed elevated 20 degrees. A set of hemodynamic pressure readings were taken. PA: 42/33; PA mean: 36; PA wedge: 32. You conclude these parameters are
    A. REFLECTIVE OF A FURTHER DECREASE IN CARDIAC FUNCTION
    B. REFLECTIVE OF PULMONARY DETERIORATION
    C. INACCURATE DUE TO THE PATIENT'S POSITION
    D. ACCURATE BECAUSE THE TRANSDUCER WAS REFERENCED CORRECTLY TO THE STERNUM

28. Select the reason you would use to support your conclusion in #27.
    A. AN INCREASE IN PA WEDGE PRESSURE MAY BE REFLECT A FURTHER DETERIORATION OF CARDIAC FUNCTION
    B. THE ELEVATION OF THE PULMONARY ARTERY SYSTOLIC AND DIASTOLIC PRESSURE IS REFLECTIVE OF PULMONARY DYSFUNCTION
    C. PULMONARY ARTERY PRESSURE MEASUREMENTS ARE NOT CONSISTENTLY REPRODUCIBLE IN THE SIDE-LYING POSITION
    D. PA PRESSURE MEASUREMENTS ARE CONSISTENTLY REPRODUCIBLE IN ALL SIDE-LYING POSITIONS, AS LONG AS THE TRANSDUCER AIR-FLUID INTERFACE IS CORRECTLY REFERENCED TO THE STERNUM

29. Your current nursing plan of care for Mr Jones, with regard to PA pressure monitoring includes
    A. PLACING HIM FLAT AND SUPINE FOR PA PRESSURE MEASUREMENTS
    B. REMOVING HIM FROM THE VENTILATOR FOR PA PRESSURE MEASUREMENTS
    C. COMPARING FLAT, SUPINE PA PRESSURE MEASUREMENTS WITH SUPINE, BACKREST UPRIGHT PRESSURE MEASUREMENTS FOR CONSISTENCY
    D. AVERAGING PA PRESSURE MEASUREMENTS OVER SEVERAL RESPIRATORY CYCLES

Thank you for your assistance with this project. Please return the answer sheet and demographic data sheet in the envelope provided. DO NOT return the test. If you would like a copy of the correct answers, rationale, and an abstract of the results of this study, please include your name and address on the form on the cover page.

Table G-1.

Answer Key

<u>Clinical Simulation: Pulmonary Artery Pressure Measurement</u>

1. B   24. D

2. B   25. C

3. C   26. C

4. D   27. C

5. D   28. C

6. C   29. C

7. C

8. B

9. C

10. B

11. B

12. B

13. C

14. C

15. C

16. D

17. D

18. C

19. D

20. D

21. D

22. C

23. C

Table G-2.

Final Test Blueprint

Clinical Simulation: Pulmonary Artery Pressure Measurement

	LEVEL I KNOWLEDGE/ COMPREHENSION	LEVEL II APPLICATION/ ANALYSIS	LEVEL III SYNTHESIS/ EVALUATION	TOTAL
CLINICAL	1,2,21,22	3,4,16,17, 19,20,23	5,6,7,8, 18	16 (55%)
TECHNICAL	9,12,13, 14,15	26,28	27,29	9 (31%)
COMPLICATIONS	10	11,25	24	4 (14%)
TOTAL	10(34%)	11(38%)	8 (28%)	29 (100%)

Table G-3.

Score Correlations

	Total	Clinical	Technical	Complications	Level 1	Level 2	Level 3
Total	1.00	0.82	0.71	0.53	0.76	0.79	0.76
Clinical	0.82	1.00	0.28	0.17	0.53	0.65	0.73
Technical	0.71	0.28	1.00	0.27	0.69	0.48	0.46
Complications	0.53	0.17	0.27	1.00	0.38	0.54	0.29
Level I	0.76	0.53	0.69	0.38	1.00	0.35	0.32
Level II	0.79	0.65	0.48	0.54	0.35	1.00	0.50
Level III	0.76	0.73	0.46	0.29	0.32	0.50	1.00

Table G-4.

Test Scores - Frequencies and Distributions

Item Number	Diff.	Disc.	Weight	Means	Frequencies		Distribution (# equals 2.5%)
1	86	0.17	A ----	16.00	9	5.0%	##
	Easy	Fair	B. 1.00	18.40	155	85.6%	###################################
			C. ----	17.60	15	8.3%	###
			D. ----	10.00	2	1.1%	
2	75	0.17	A. ----	16.00	11	6.1%	##
	Medium	Fair	B. 1.00	18.64	135	74.6%	##############################
			C. ----	17.30	23	12.7%	#####
			D. ----	17.18	11	6.1%	##
			Omits	21.00	1	0.6%	
3	94	0.20	A. ----	-----	0	0.0%	
	Easy	Fair	B. ----	8.00	1	0.6%	
			C. 1.00	18.23	171	94.5%	#######################################
			D. ----	15.44	9	5.0%	##
4	96	0.13	A. ----	16.67	4	2.2%	#
	Easy	Fair	B. ----	14.00	3	1.7%	#
			C. ----	-----	0	0.0%	
			D. 1.00	18.13	173	95.6%	#######################################
			Omits	16.00	1	0.6%	
5	67	0.20	A. ----	13.00	3	1.7%	#
	Medium	Fair	B. ----	17.97	31	17.1%	#######
			C. ----	16.69	26	14.4%	######
			D. 1.00	18.88	121	66.9%	###########################
6	71	0.20	A. ----	19.00	2	1.1%	
	Medium	Fair	B. ----	17.02	45	24.9%	##########
			C. 1.00	18.78	128	70.7%	#############################
			D. ----	16.50	6	3.3%	#
7	66	0.27	A. ----	17.18	17	9.4%	####
	Medium	Fair	B. ----	15.00	15	8.3%	###
			C. 1.00	19.09	119	65.7%	##########################
			D. ----	17.52	29	16.0%	######
			Omits	19.00	1	0.6%	

Item Number	Diff.	Disc.	Weight	Means	Frequencies		Distribution (# equals 2.5%)
8	81	0.14	A. ----	17.36	25	13.8%	######
	Medium	Fair	B. 1.00	18.44	147.	81.2	#################################
			C. ----	13.75	4	2.2%	#
			D. ----	17.60	5	2.8%	#
9	22	0.05	A. ----	18.70	139	76.8%	###############################
	Hard	Poor	B. ----	15.00	1	0.6%	
			C. 1.00	19.15	39	21.5%	#########
			D. ----	17,50	2	1.1%	
10	67	0.23	A. ----	18.00	6	3.3%	#
	Medium	Fair	B. 1.00	18.94	121	66.9	##########################
			C. ----	17.48	21	11.6%	#####
			D. -----	16.84	31	17.1%	#######
			Omits	12.50	2	1.1%	
11	43	0.18	A. ----	19.57	44	24.3%	##########
	Hard	Fair	B. 1.00	19.39	77	42.5%	#################
			C. ----	15.30	10	5.5%	##
			D. ----	17.27	49	27.1%	###########
			Omits	5.00	1	0.6%	
12	57	0.36	A. ----	17.62	61	33.7%	#############
	Medium	Good	B. 1.00	19.60	104	57.5%	#######################
			C. ----	10.50	2	1.1%	
			D. ----	15.17	12	6.6%	###
			Omits	7.50%	2	1.1%	
13	61	0.26	A. ----	12.83	6	3.3%	#
	Medium	Fair	B. ----	18.48	25	13.8%	######
			C. 1.00	19.18	111	61.3%	########################
			D. ----	17.32	37	20.4%	########
			Omits	7.50	2	1.1%	
14	27	0.37	A. ----	17.96	49	27.1%	###########
	Hard	Good	B. ----	18.95	63	34.8%	#############
			C. 1.00	21.06	49	27.1%	###########
			D. ----	14.82	17	9.4%	####
			Omits	9.33	3	1.7%	
15	55	0.13	A. ----	13.00	1	0.6%	
	Medium	Fair	B. ----	18.57	54	29.8%	###########
			C. 1.00	18.88	100	55.2	######################
			D. ----	17.22	23	12.7%	#####
			Omits	11.67	3	1.7%	#

Item Number	Diff.	Disc.	Weight	Means	Frequencies		Distribution (# equals 2.5%)
16	86	0.18	A. ----	14.57	7	3.9%	##
	Easy	Fair	B. ----	18.29	14	7.7%	###
			C. ----	17.00	2	1.1%	
			D. 1.00	18.41	156	86.2%	###################################
			Omits	7.50	2	1.1%	
17	92	0.20	A. ----	-----	0	0.0%	
	Easy	Fair	B. ----	18.00	6	3.3%	#
			C. ----	15.43	7	3.9%	##
			D. 1.00	18.30	166	91.7%	####################################
			Omits	7.50	2	1.1%	
18	58	0.38	A. ----	16.07	14	7.7%	###
	Medium	Good	B. ----	17.37	16	8.8%	####
			C. 1.00	19.67	105	58.0%	#######################
			D. ----	16.98	44	24.3%	##########
			Omits	7.50	2	1.1%	
19	61	0.28	A. ----	15.87	15	8.3%	###
	Medium	Fair	B. ----	19.50	2	1.1%	
			C. ----	17.65	49	27.1%	###########
			D. 1.00	19.25	111	61.3%	#########################
			Omits	11.25	4	2.2%	#
20	62	0.31	A. ----	17.30	10	5.5%	##
	Medium	Good	B. ----	16.73	41	22.7%	#########
			C. ----	17,87	15	8.3%	###
			D. 1.00	19.30	112	61.9%	#########################
			Omits	11.33	3	1.7%	#
21	78	0.29	A. ----	16.48	29	16.0%	######
	Medium	Fair	B. ----	12,67	3	1.7%	#
			C. ----	17.67	3	1.7%	#
			D. 1.00	18.80	142	78.5%	################################
			Omits	13.50	4	2.2%	#
22	54	0.25	A. ----	14.67	3	1.7%	#
	Medium	Fair	B. ----	17.52	71	39.2%	################
			C. 1.00	19.34	98	54.1%	######################
			D. ----	19.86	7	3.9%	##
			Omits	7.50	2	1.1%	
23	83	0.34	A. ----	16.00	3	1.7%	#
	Medium	Good	B. ----	16.72	18	9.9%	####
			C. 1.00	18.76	150	82.9%	#######################################
			D. ----	13.80	5	2.6%	#
			Omits	10.60	5	2.6%	#

Item Number	Diff.	Disc.	Weight	Means	Frequencies		Distribution (# equals 2.5%)
24	83	0.33	A. ----	17.18	11	6.1%	##
	Medium	Good	B. ----	17.12	8	4.4%	##
			C. ----	13.67	6	3.3%	#
			D. 1.00	18.73	151	83.4%	###################################
			Omits	9.60	5	2.8%	#
25	68	0.27	A. ----	17.91	33	18.2%	#######
	Medium	Fair	B. ----	17.23	13	7.2%	###
			C. 1.00	19.02	123	68.0%	###########################
			D. ----	15.57	7	3.9%	##
			Omits	9.60	5	2.8%	#
26	34	0.14	A. ----	16.69	13	7.2%	###
	Hard	Fair	B. ----	19.29	51	28.2%	###########
			C. 1.00	19.41	61	33.7%	#############
			D. ----	18.45	51	28.2%	###########
			Omits	9.60	5	2.8%	#
27	65	0.16	A. ----	17.27	15	8.3%	###
	Medium	Fair	B. ----	20.00	2	1.1%	
			C. 1.00	18.78	118	65.2%	##########################
			D. ----	18.19	42	23.2%	#########
			Omits	9.50	4	2.2%	#
28	61	0.18	A. ----	18.27	11	6.1%	##
	Medium	Fair	B. ----	18.50	2	1.1%	
			C. 1.00	18.95	110	60.8%	########################
			D. ----	18.00	53	29.3%	############
			Omits	9.60	5	2.8%	#
29	45	0.16	A. ----	18.58	53	29.3%	############
	Hard	Fair	B. ----	15.25	4	2.2%	#
			C. 1.00	19.24	82	45.3%	##################
			D. ----	18.63	35	19.3%	########
			Omits	11.00	7	3.9%	##

Diff = Difficulty
Disc = Discrimination

APPENDIX H

Demographic Data Sheet

Demographic Data Sheet

Please fill in the blank or circle the letter corresponding with the answer that is most accurate as of this date

1 Gender
   A MALE
   B FEMALE

2 Age (specify in years) _____

3 State of Residence _____

4 Basic Education in Nursing
   A DIPLOMA
   B ASSOCIATE DEGREE
   C BACCALAUREATE OR HIGHER
   D OTHER _____

5 Highest Nursing Degree Held
   A DIPLOMA
   B ASSOCIATE DEGREE
   C BACCALAUREATE
   D MASTERS
   E DOCTORATE
   F OTHER _____

6 Additional Certification (circle ALL that are appropriate)
   A ACLS PROVIDER
   B ACLS INSTRUCTOR
   C ACLS AFFILIATE FACULTY
   D CCRN
   E OTHER _____

7 Years of Nursing Experience
   A LESS THAN 2 YEARS
   B 2 TO 3 YEARS
   C 4 TO 5 YEARS
   D 6 TO 10 YEARS
   E 11 TO 15 YEARS
   F 16 TO 20 YEARS
   G 21 + YEARS

8 Years of Critical Care Experience
   A LESS THAN 2 YEARS
   B 2 TO 3 YEARS
   C 4 TO 5 YEARS
   D 6 TO 10 YEARS
   E 11 TO 15 YEARS
   F 16 TO 20 YEARS
   G 21 + YEARS

9 Years SINCE Active in Critical Care
   A LESS THAN 2 YEARS
   B 2 TO 3 YEARS
   C 4 TO 5 YEARS
   D 6 TO 10 YEARS
   E 11 TO 15 YEARS
   F 16 TO 20 YEARS
   G 21 + YEARS
   H NOT APPLICABLE

10 Employed in Nursing
   A FULL TIME
   B PART TIME
   C NOT EMPLOYED IN NURSING
   D RETIRED
   E FULL TIME STUDENT

11 Type of facility in which primarily employed (CHECK ONE)
   A COMMUNITY HOSPITAL
   B UNIVERSITY AFFILIATED MEDICAL CENTER
   C MILITARY
   D FEDERAL HOSPITAL
   E STATE HOSPITAL
   F COUNTY HOSPITAL
   G ACADEMIC INSTITUTION (FACULTY)
   H ACADEMIC INSTITUTION (STUDENT)
   I SELF EMPLOYED
   J REGISTRY
   K PRIVATE INDUSTRY
   L OTHER _____

12 Hospital Size (number of beds)
   A LESS THAN 50
   B 50 TO 99
   C 100 TO 199
   D 200 TO 299
   E 300 TO 399
   F 400 +
   G NOT APPLICABLE

13 Primary Area of Employment (CHOOSE ONE)
   A COMBINED ICU/CCU
   B INTENSIVE CARE UNIT (ICU)
   C CORONARY CARE UNIT
   D MEDICAL ICU
   E SURGICAL ICU
   F RESPIRATORY ICU
   G CARDIOVASCULAR-SURGICAL ICU
   H NEURO/NEURO-SURGICAL ICU
   I TRAUMA ICU
   J EMERGENCY ROOM
   K BURN ICU
   L RECOVERY ROOM
   M ROTATE CRITICAL CARE AREAS
   N STUDENT
   O OTHER _____

14 Present Position
   A STAFF OR GENERAL DUTY
   B CHARGE NURSE/TEAM LEADER
   C HEAD NURSE/ASST. HEAD NURSE
   D NURSING ADMINISTRATOR
   E SUPERVISOR/ASST. SUPERVISOR
   F INSERVICE/STAFF DEVELOPMENT
   G ACADEMIC INSTRUCTOR
   H CLINICAL NURSE SPECIALIST/NURSE CLINICIAN
   I NURSE PRACTITIONER
   J FULL TIME STUDENT
   K OTHER _____

15 Describe your level of knowledge and ability to utilize information related to pulmonary artery pressure measurement
   A NOVICE
   B ADVANCED BEGINNER
   C COMPETENT
   D PROFICIENT
   E EXPERT

APPENDIX I

Cover Letter

UNIVERSITY OF WASHINGTON

SCHOOL OF NURSING

D E P A R T M E N T   O F   P H Y S I O L O G I C A L   N U R S I N G

Dear AACN Member,

I am a member of AACN and a graduate student in the Department of Physiological Nursing at the University of Washington. In partial fulfillment of my program requirements, I am surveying critical care nurses' knowledge of pulmonary artery pressure measurement and ability to utilize this knowledge in a clinical scenario. It is hoped through this study to identify our knowledge of pulmonary artery pressure measurement and specific educational needs. Because this study can benefit our practice as critical care nurses, I ask you to consider participation. You DO NOT have to have current experience in critical care to complete this study. Your input, regardless of experience level will provide useful information related to the educational needs of all critical care nurses.

Your name was randomly selected by AACN from the AACN membership list and materials were mailed from a mailing house. The demographic survey and sensor mark answer sheets were number coded prior to forwarding to the mailing house so the investigator has no record of the member to whom the surveys were sent.

Participation is voluntary; you are free to refuse to participate without penalty. Consent to participate will be implied by the return of the completed answer and demographic sheet. In order to ensure anonymity, please do not write your name or address on the answer sheet or demographic data sheet. Survey data will be identified by number only, therefore, there is no risk of professional consequence to you as a result of the questionnaire scores. The answer sheet and demographic data sheets will be secured indefinitely. Only members of the research team will have access to the data. There is no direct benefit to you for completion of this questionnaire, however, the development of a reliable and valid tool, and its use to evaluate critical care nurses' knowledge of pulmonary artery pressure measurement will facilitate the development of specific instructional programs. The results of this study may be used for thesis development, professional publication and professional meetings. If you wish to receive an abstract and answer sheet please complete the data information sheet on the last page of the test. This personal information will be separated from the test prior to scoring.

Enclosed you will find a questionnaire regarding pulmonary artery pressure measurement, a marked sense answer sheet, and a demographic data sheet. The demographic data sheet and questionnaire will take approximately 30 minutes to complete. Please return the mark sense answer sheet and demographic data sheet in the enclosed, postage paid envelope within two weeks. I would request that you not use any references or consult with anyone to complete the questionnaire, as this will alter the validity of the results.

I may be contacted through the Department of Physiological Nursing, University of Washington (Phone: 206-685-2266), and I would be happy to answer any questions you may have. I thank you again for your participation, and commend you for you commitment to critical care research.

Sincerely,

Elizabeth J Bridges

ELIZABETH J. BRIDGES, RN, BSN, CCRN
Graduate Student, Department of Physiological Nursing
University of Washington

Elizabeth J. Bridges is enrolled in the Department of Physiological Nursing, at the University of Washington. Your assistance with this project is greatly appreciated.

Susan L. Woods

SUSAN L. WOODS, RN, PhD
Faculty Advisor, Associate Professor
Department Of Physiological Nursing
University of Washington

APPENDIX J

Follow-Up Postcard

Dear AACN Member:

You should have received a questionnaire related to pulmonary artery pressure monitoring in the mail approximately one week ago. If you have already returned your questionnaire answer sheet and demographic data sheet, I thank you for your promptness. If you have not yet had a chance to complete the questionnaire, please do so and return the answer sheet and demographic data sheet in the envelope enclosed in the survey packet. Again, thank you for your interest in critical care nursing research.

Sincerely,

Elizabeth Bridges, RN, BSN, CCRN
Department of Physiological Nursing, SM-28
University of Washington, Seattle, WA 98195
(206) 685-2266

APPENDIX K

Letter of Support

American Association of Critical Care Nurses

AACN

AMERICAN ASSOCIATION
OF CRITICAL-CARE NURSES

May 6, 1991

President
 S Ann Evans
 RN MS MBA CNAA

President-Elect
 Wanda Roberts Johnson
 RN MN

Treasurer
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 Cindy Sorensen
 RN BSN CCRN

 Nancy A Urban
 RN MSN CCRN

 Jean M Vestlin-Castro
 RN MSN CCRN CS

 Pamela Moopor Wolff
 RN MSN CCRN

 Kimberly Woods-McCormick
 RN BSN CCRN

Elizabeth J. Bridges, RN, BSN, CCRN
5844 NE 75th, #A313
Seattle, WA 98115

Dear Elizabeth:

Your request for use of the AACN mailing list for research is
approved. The label request has been forwarded to the Computer
Department for processing.

Labels will be mailed to your mailing house within four weeks.
The mailing house is instructed not to release the list or labeled mail
directly to you under any circumstances.

Best wishes with your research. If you have any questions about
the mailing labels, call the Department of Research at (714) 362-2000.

Sincerely,

Karen R. Sechrist, PhD, RN
Director of Research

Executive Director
 Sarah Sanford
 RN MA CNAA

101 Columbia, Aliso Viejo, CA 92656
714-362-2000
800-899-AACN
FAX 714-362-2020
TLX 299917 AACN UR

APPENDIX L

Correct Answers and Rationale for

Clinical Simulation: Pulmonary Artery Pressure Measurement

Item 1. (B). Content: clinical. Cognitive Level: 1.
Preload refers to end-diastolic myocardial fiber length, which in the intact ventricle is related to the end-diastolic volume. Based on the Frank-Starling law of the heart, an increase in fiber length, within limits, is associated with an increase in force of myocardial contraction. This relationship can be manipulated clinically to increase cardiac output.

Item 2. (B). Content: clinical. Cognitive Level: 1
Afterload is the wall tension that is developed during ventricular systole, and is related to the force opposing ventricular ejection. Clinically, systemic vascular resistance (SVR) is used as an indicator of left ventricular afterload. Systemic vascular resistance is calculated from data provided by the pulmonary artery (PA) catheter system.

$$SVR = \frac{[(MAP - RAP \text{ (mm Hg)}] \text{ X } 80}{\text{Cardiac output}} = \text{dynes/sec/cm}^{-5}$$

Where:
MAP = mean arterial pressure
$$MAP = \frac{\text{(systolic blood pressure} + 2 \text{ (diastolic blood pressure)}}{3}$$
RAP = right atrial pressure

Normal SVR = 900 to 1300 dynes/sec/cm^{-5}

Item 3. (C). Content: clinical. Cognitive level 2.
There is a clinical relationship between the PA wedge pressure and the presence or absence of pulmonary congestion, and between cardiac index and the presence or absence of systemic hypoperfusion. In the presence of an acute myocardial infarction a PA wedge pressure greater than 18 torr (1 torr = 1 mm Hg) is associated with the onset of pulmonary congestion, and a cardiac index of less than 2.2 liter/min/m^2 with systemic hypoperfusion.

The clinical findings for the patient in this case are pulmonary edema without systemic hypoperfusion.

Item 4. (D). Content: clinical. Cognitive Level: 3.
   As noted in Table 1 individuals can be classified into clinical subsets on the basis of their PA wedge
   pressure and cardiac index.

Table 1.

Hemodynamic Subsets. From "Medical Therapy of Acute Myocardial Infarction by Application of Hemodynamic
Subsets" by J.S. Forrester, G. Diamond, G. Chatterjee, H.J.C. Swan, 1976, New England Journal of Medicine,
295(24), p. 1361. Copyright 1976 by the New England Journal of Medicine. Adapted by permission. PAWP =
pulmonary artery wedge pressure; CI = cardiac index.

SUBSET	PAWP 18 mm	CI > 2.2 l/min/m²	PULMONARY CONGESTION	PERIPHERAL HYPOPERFUSION	MORTALITY
I	NO	NO	NO	NO	1-3%
II	YES	NO	YES	NO	9-9%
III	NO	YES	NO	YES	2-22%
IV	YES	YES	YES	YES	9-60%

Item 5. (D). Content: clinical. Cognitive Level: 3.
   Treatment of patient s with a myocardial infarction is based on the relationship between pulmonary artery
   wedge pressure and pulmonary congestion, and between cardiac index and systemic perfusion (Forrester et
   al, 1976). These relationships can be viewed graphically by the creation of a ventricular function curve.
   (Figure 1).
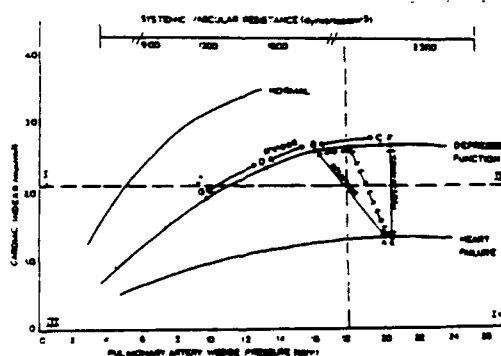
Figure 1. Ventricular function curve and hemodynamic subsets.

   As noted in Figure 1, a reduction in preload (Point C to Point B) will result in reduction in PA wedge
   pressure without a marked decreased in cardiac index. However, excessive preload reduction may result in
   a decrease in cardiac index (Point G). An increase in afterload will move the curve down and to the right
   (Point B to Point A), and a decrease in afterload will move the curve up and to the right (Point A to
   Point B). An increase in contractility, independent of changes in preload or afterload will move the
   curve up (Point E to Point F), while a decrease in contractility moves the curve down (Point F to Point
   E).

Creation of a ventricular function curve for Mr Jones will allow us to follow his progress graphically (Figure 2). In this case, the cardiac index of 2.4 L/min/m^2 precludes the need for therapy to increase contractility, and the systemic vascular resistance of 1088 dynes/sec/cm^{-5} is normal; therefore afterload reduction is not warranted. If the systemic vascular resistance were elevated, consideration of vasodilator therapy would be appropriate.
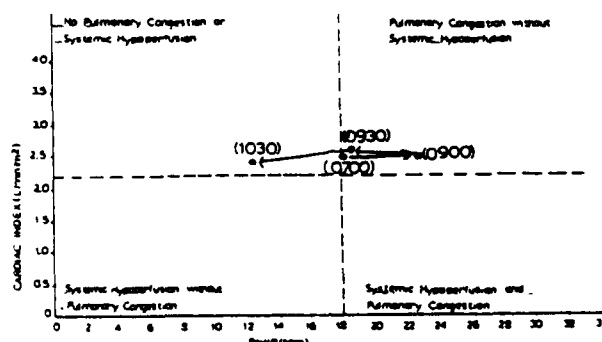

Figure 2. Ventricular function curve to track Mr Jones's hemodynamic status.

Item 6. (C) Content: clinical. Cognitive Level: 3.
There are several methods for preload reduction. Diuretic therapy is generally used to meet this clinical goal. If the patient has systemic hypertension, a peripheral vasodilator (nitroglycerin, nitroprusside) may be effective in reducing the pulmonary artery wedge pressure without reducing cardiac index. In the case of systemic hypertension the vasodilator would reduce both preload and afterload.

Item 7. (C). Content: clinical. Cognitive Level: 3.
There was a reduction in PA pressure from 38/29 torr to 34/25 torr, PA mean pressure from 33 to 28, and PA wedge pressure from 23 to 19. There was no change in cardiac index (Figure 2, 0900 to 0930).
An increase in contractility would improve the ejection of blood from the ventricle, with a subsequent reduction in preload (Figure 1, Point E to Point F). A decrease in afterload would also result in improved force of contraction (less resistance to ejection), and the subsequent reduction in end-diastolic volume (Figure 1, Point A to Point B). Clinically significant changes in PA pressure are described as 4 torr for PA diastolic and PA wedge pressure, and 5 torr for PA systolic and mean PA pressures. Any pressure change less than these values may simply reflect a normal fluctuation in pressure. An increase in preload would result in an increase in PA wedge pressure (Figure 1, Point B to Point C), and exacerbation of the pulmonary congestion.

Item 8. (B). Content: clinical. Cognitive Level: 3.
Changes in PA wedge pressure occur 1 to 24 hours before changes in the clinical signs of left ventricular failure. Therefore, the PA catheter data provides a more timely indicator of the change in pulmonary status, and prevents overtreatment. While a PA wedge pressure of 12 torr may indicate inadequate end-diastolic volume in a patient with a myocardial infarction, it is not indicative of "severe" hypovolemia.

Item 9. (C). Content: technical. Cognitive Level 1.
The phlebostatic axis is the intersection of a frontal plane passing through one-half the anterior posterior diameter and a plane that transects the body at the fourth intercostal space at the sternal margin. Use of the mid-axillary line as opposed to one-half the anterior-posterior diameter may result in a pressure difference of up to 6 torr.

Item 10. (B). Content: complication. Cognitive Level: 1.

This waveform (Figure 3) is consistent with a PA wedge pressure tracing. During continuous PA pressure monitoring the presence of a PA wedge waveform is indicative of distal migration of the catheter, and wedging in a small pulmonary vessel. Pericardial tamponade is characterized by an increase and equalization of all pressures, a right atrial pressure tracing with a deep "x" descent, and a brief "y" descent. In addition, the right atrial pressure decreases during inspiration. Acute mitral insufficiency is characterized by a giant "V" wave on the PA and PA wedge pressure tracing. The PA end-diastolic pressure may be less than the PA wedge pressure due to the presence of the huge "V" wave. However, the presence of a large "V" wave can also be caused by an acute ventricular septal defect.
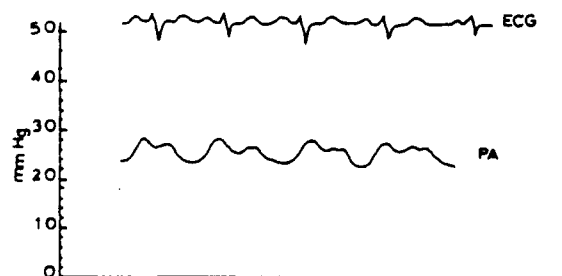
Figure 3. Spontaneous wedge.

Item 11. (B). Content: complications. Cognitive Level: 2.

The first nursing action is to attempt to dislodge the catheter passively. While gentle flushing of the catheter system may be considered, administration of high pressure or inflation of the balloon in a small pulmonary vessel may result in the catastrophic rupture of the vessel. Although this PA wedge pressure is consistent with left ventricular failure requiring preload reduction, in this situation the presence of a spontaneous wedge is a potentially serious complication, and requires immediate treatment.

Item 12. (B) Content: technical. Cognitive Level: 1.

The changes in pleural pressure that occur during the respiratory cycle are transmitted to cardiovascular structures in the thorax, and are reflected by changes in PA pressure measurements during inspiration and expiration. During spontaneous ventilation, PA pressures _decrease_ during inspiration, and _increase_ during expiration. During mechanical ventilation PA pressures _increase_ during inspiration, due to positive-pressure ventilation, and _decrease_ during expiration. At end-expiration pleural pressure equals atmospheric pressure, and no air flow occurs. Therefore, measurements of PA pressures at end-expiration are not affected by pleural pressure changes, and provide the most accurate indication of PA pressures (Figure 4). The normal PA systolic is 15 to 30 torr.
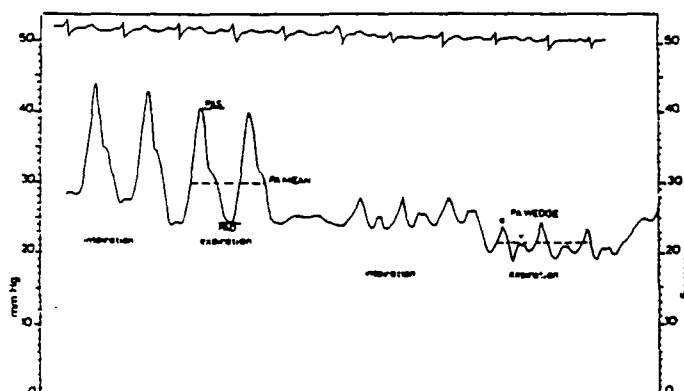
Figure 4. Pulmonary artery and PA wedge pressure measurement at end-expiration in a mechanically ventilated patient.

Item 13 (C). Content: technical. Cognitive Level: 1.

The PA diastolic pressure is measured immediately before systole (upstroke of the waveform), and normally ranges from 3 to 12 torr. As noted in item 12, PA diastolic is read at end-expiration (Figure 4).

Item 14 (C). Content: technical. Cognitive Level: 1.

Mean PA pressure normally ranges from 9 to 16 torr, and is measured by bisecting the end-expiratory pressure waveform so there are equal areas above and below the bisection. The point of the bisection is not necessarily consistent with the dicrotic notch (Figure 4).

Item 15. (C). Content: technical. Cognitive Level: 1.

The PA wedge pressure tracing consists of two positive deflections. The "a" wave reflects left atrial systole, and follows the P-R interval on the ECG. The "v" wave reflects left ventricular systole, and the peak of the "v" wave occurs during the T-P interval on the ECG. Note in this tracing that the "a" wave is peaked due to a decrease in left ventricular compliance. The decrease in compliance causes pressure in the left atria to rise, as the atrium ejects blood into the stiff ventricle. The PA wedge pressure is a mean pressure, and is measured by bisecting the "a" and "v" waves so there is an equal area above and below the bisection. The measurements are made on the portion of the waveform that represents end-expiration. A normal PA wedge is 4 to 12 torr (Figure 4).

Item 16. (D). Content: clinical. Cognitive Level: 2.

The hemodynamic parameters in items 12 through 16 are consistent with the diagnosis of left ventricular failure. Left ventricular failure is characterized by a normal or increased right atrial pressure, and increased PA and PA wedge pressure. If there is no pulmonary pathology the PA end-diastolic - PA wedge pressure gradient will be less than 6 torr.

Acute pericardial tamponade is characterized by equalization of pressures. The right atrial pressure tracing will have a deep "x" descent, and a brief "y" descent. The mean right atrial pressure decreases during inspiration (negative Kussmaul's sign), which differentiates pericardial tamponade from other pathologies that cause equalization of heart pressures (right ventricular infarction and pericardial constriction).

Mitral insufficiency is characterized by a giant "V" wave inscribed on the PA and PA wedge pressure tracing. The PA end-diastolic pressure may be less than the PA wedge pressure due to the huge "V" wave. However, the presence of a large "V" wave is neither sensitive nor specific to mitral regurgitation, and can also be caused by an acute ventricular septal defect.

Pulmonary embolus is characterized by an increase in right atrial and PA pressures. The PA wedge pressure may be normal or decreased secondary to a decrease in left ventricular preload. The right atrial waveform has a deep "v" wave with a steep "y" descent due to tricuspid regurgitation. The PA end-diastolic - PA wedge pressure gradient will be greater than 6 torr, indicating pulmonary pathology.

Item 17. (D) Content: clinical. Cognitive Level: 3.

A cardiac index of less than 2.2 L/min/m^2 is associated with systemic hypoperfusion, and a PA wedge pressure of greater than 18 torr is associated with pulmonary congestion. These hemodynamic parameters are consistent with Forrester's (1976) subset IV, and are clinically indicative of cardiogenic shock (Table 1).

Item 18. (C). Content: clinical. Cognitive Level: 3.

One therapeutic option for patients with cardiogenic shock is the reduction of preload (decrease in PA wedge pressure). Vasodilator or diuretic therapy is recommended to achieve this goal (Figure 5, Point X to Point A). Care must be taken to avoid excessive preload reduction that may result in hypotension and a further decrease in cardiac index (Point B).

In this case, volume augmentation would exacerbate the patient's condition (Point C). Vasoconstriction, with the subsequent increase in afterload would result in further myocardial dysfunction secondary to an increase in myocardial oxygen consumption (Point D). Chronotropic therapy would not be warrranted in this situation, where the heart rate was already accelerated.
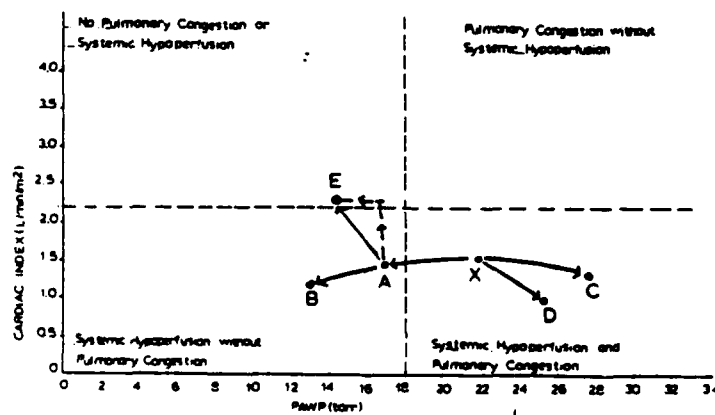

Figure 5. Ventricular function curve graphically demonstrating the effects of various therapies on cardiac index and PA wedge pressure in a patient in cardiogenic shock. (See text).

Item 19. (D). Content: clinical. Cognitive Level: 3.
The goal of therapy is a reduction in preload. The reduction in the PA wedge pressure should relieve pulmonary congestion. While an increase in contractility is also a therapeutic option for treatment of cardiogenic shock, there was no option in item 18 related to contractility.

Item 20. (D). Content: clinical. Cognitive Level: 2.
The other therapeutic goal for cardiogenic shock is the increase in cardiac index. Afterload reduction, by use of a vasodilator (nitroprusside) or mechanical assist device (inta-aortic balloon pump), with a subsequent increase in cardiac index and a reduction in PA wedge pressure (Figure 5, Point X to Point E). Because vasodilator therapy has the potential for exacerbating hypotension, an inotropic agent (dopamine) is often added to maintain systemic blood pressure.

Item 21. (D). Content: clinical. Cognitive Level: 1.
As noted in Figure 5, a ventricular function curve provides a graphic representation of Starling's law of the heart, i.e., an increase in fiber length (end-diastolic volume), within limits, will increase force of contraction and cardiac output. Indices of left ventricular end-diastolic volume include: pulmonary artery wedge pressure (PAWP), PA diastolic pressure, and left ventricular end-diastolic pressure. Central venous pressure is poorly correlated with left-sided heart function, particularly in patients with impaired myocardial function.

Item 22. (C). Content area: clinical. Cognitive Level: 1.
Cardiac work or ventricular performance are most often measured by stroke volume, stroke volume index, stroke work, stroke work index, cardiac output, or cardiac index. Use of stroke volume controls for the independent effect of heart rate on cardiac output. As noted in item 21, PA wedge pressure is an indicator of end-diastolic volume.

Item 23. (C). Content area: clinical. Cognitive Level: 2.
The ideal therapeutic outcome of the treatment for cardiogenic shock is a decrease in preload, and an increase in cardiac index (Figure 6, Point X to Point C). Point A reflects a further increase in preload (volume augmentation), and exacerbation of the pulmonary congestion. Point B reflects an increase in contractility and cardiac index, without a decrease in preload. Point D reflects the effect of vasoconstriction (increased afterload), with a further increase in preload and a decrease in cardiac index.
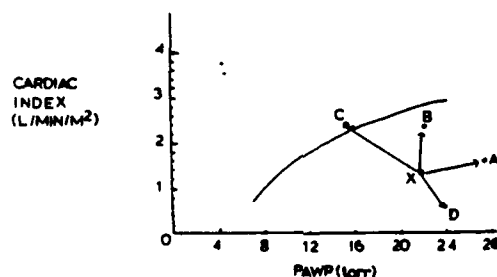
Figure 6. Effect of therapy for cardiogenic shock.

Item 24. (D). Content: complications. Cognitive Level: 2.

The waveform tracing is consistent with withdrawal of the PA catheter tip into the right ventricle. Characteristics of a right ventricular tracing include similarity between right ventricular diastolic pressure and right atrial mean pressure, and similarity between right ventricular systolic pressure and PA systolic pressure. The PA tracing can be differentiated from the right ventricular tracing by a diastolic pressure that is greater than the mean right atrial pressure. In addition, the presence of ectopy from the right ventricle is consistent with endocardial irritation from the catheter tip.
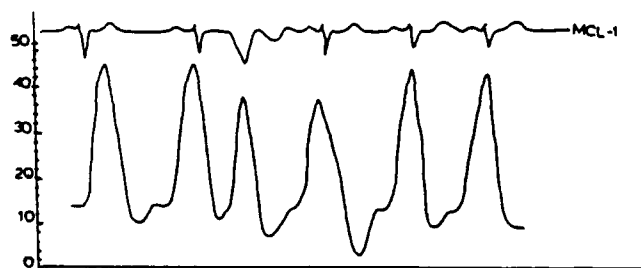

Figure 7. Right ventricular pressure tracing.

Item 25. (C). Content: complications. Cognitive Level: 3.

Inflation of the PA catheter balloon will decrease the impact of the catheter tip on the endocardium. Withdrawal of the catheter into the right atrium is also an option for catheter-induced ectopy.

Item 26 (C). Content: technical. Cognitive Level: 2.

Left ventricular distention is caused by the difference between intravascular pressure (PA wedge pressure) and extravascular (intravascular) pressure. Generally, intrapleural pressure is considered to be atmospheric; however, in the presence of positive end-expiratory pressure (PEEP), intrapleural pressure is increased. To correct for the effect of PEEP, one-half of the applied PEEP increment (in torr) is subtracted from the PA wedge pressure. In item 26:
(1) Convert the applied PEEP increment from cm H_2O to torr
10 cm H_2O/(1.36 cm H_2O/torr) = 7.35 torr.
(2) Subtract one-half the PEEP increment from the measured PA wedge pressure.
30 torr - 7.35 torr/2 = 26 torr.

Use of this formula allows for an approximation of the "true" intravascular pressure.

Item 27. (C). Content: technical. Cognitive Level: 3. Current research indicates that the only lateral position that has been shown to provide reliable PA and PA wedge pressures relative to the flat, supine position is 90-degrees lateral with 0-degree backrest elevation. The reference for the left lateral decubitus position is the fourth intercostal space, left parasternal border, and the fourth intercostal space at the mid-sternum for the right lateral decubitus position.

Item 28. (C). Content: technical. Cognitive Level: 2.
See item 27.

Item 29. *Measurement of all pressures in the flat, supine position may be poorly tolerated* by patients with
cardiovascular or pulmonary compromise. There are numerous research studies that indicate that PA and PA
wedge pressures can be attained in the supine position with backrest elevation up to 60 degrees. However,
individual responses to position changes occur; therefore, pressure measurements in the supine, backrest-
elevated position should be compared with flat, supine measurements to determine consistency.

Averaging PA pressure measurements over several respiratory cycles results in error due to ventilatory
effects (see Item 12). However, it is recommended to average several end-expiratory pressures when
measuring hemodynamic parameters.

There is no significant difference between pressures measured end-expiration on mechanical ventilation
without positive end-expiratory pressure (PEEP) versus off the ventilator. Removal of the patient from
the ventilator for hemodynamic pressure measurement does not provide an accurate description of the true
cardiopulmonary pressures that the pateitn is subjected to as a result of positive pressure ventilation.
In addition, in patients with left ventricular dysfunction, removal of the patient from the ventilator may
cause rebound hypervolemia and further left ventricular dysfunction.

Scoring of the Test

Each test item has been categorized into two area:

1) Content:

    A) Clinical: refers to the clinical management of the patient, including diagnosis, therapeutic actions, and goal setting.

    B) Technical: refers to issues that affect the reliability and validity of PA pressure measurement, e.g., positioning, ventilatory effect, referencing the system, reading the analog waveforms.

    C) Complications: refers to the recognition and management of complications associated with PA pressure measurement, e.g., right ventricular catheter placement, spontaneous wedge.

2) Cognitive levels are consistent with Bloom's (1956) taxonomy. The cognitive levels are grouped in a manner consistent with the CCRN examination.

    A) Level 1 - knowledge and comprehension

        Knowledge: the knowing of facts and specific bits of information

        Comprehension: the ability to interpret, translate, and extrapolate specific bits of information.

    B) Level 2 - application and analysis

        Application: the ability to use an abstraction in a situation where its use is not specified.

        Analysis: the ability to breakdown material into its constituent parts, and detection of the relationships of the parts and the way they are organized.

    C) Level 3 - synthesis and evaluation

        Synthesis: the process of working with elements, and combining them in such a way as to constitute a pattern not clearly there before.

        Evaluation: the ability to make judgements about the value of ideas, solutions, and ideas.

To score the test, use the attached test blueprint. Subset scores are the total correct divided by the number of items in the subset. Use of this scoring method will allow for the specific identification of learning needs.

Blueprint

Clinical Simulation: Pulmonary Artery Pressure Measurement

	LEVEL I KNOWLEDGE/ COMPREHENSION	LEVEL II APPLICATION/ ANALYSIS	LEVEL III SYNTHESIS/ EVALUATION	TOTAL
CLINICAL	1,2,21,22	3,4,16,17, 19,20,23	5,6,7,8, 18	16
TECHNICAL	9,12,13, 14,15	26,28	27,29	9
COMPLICATIONS	10	11,25	24	4
TOTAL	10	11	8	29

Key: Numbers refer to the corresponding test item.

APPENDIX M

Human Subjects Documentation

University procedures provide for departmental review of research involving human subjects if that research is exempt from Federal regulations. The exempt categories are described on the back of this form. Exempt research may be approved by the Department Chair, Director, or Dean provided it is in accord with the general principles stated in the UW Handbook, Vol. IV, Part II, Ch. 2, Sect. 1. (see back of form). This form, properly endorsed, certifies that the research described here qualifies for exemption.

PRINCIPAL INVESTIGATOR __Elizabeth J Bridges, RN, BSN__ ACADEMIC TITLE __Graduate Student__

FACULTY SPONSOR (IF PRINCIPAL INVESTIGATOR IS A STUDENT) CCRN __Susan L Woods, RN, PhD, Associate Profess__

DEPARTMENT __Physiological Nursing__ MAIL STOP __SM-28__ TELEPHONE __685-2266__

PROJECT TITLE __Evaluation of Critical Care Nurses' Knowledge and Ability to Utiliz Information Related to Pulmonary Artery Pressure Measurement__

STARTING DATE __15 Mar 1991__ ANTICIPATED TERMINATION DATE __23 Aug 1991__

GRANT TITLE (IF DIFFERENT FROM PROJECT TITLE) _____

PRINCIPAL INVESTIGATOR ON GRANT (IF DIFFERENT FROM PI LISTED ABOVE) _____

FUNDING AGENCY AND APPLICATION DUE DATE (IF APPLICABLE) _____

I. Under which category does this research qualify for exemption? See the back for a description of exempt categories. Check one. ___1 _X_2 ___3 ___4 ___5 ___6

II. ABSTRACT: State briefly (limit 500 words), a) the purpose(s) of the research, b) what subjects will do (if applicable), and c) the nature of the data to be obtained. The purpose of this study is to evaluate critical care nurses' knowledge and ability to utilize information related to pulmonary arter pressure measurement, and the extent that relevant demographic data (ag gender, experience in nursing, experience in critical care, hospital si area of employment, basic and advanced education, and additional certification) correlate with the mastery of this information. Procedure: One thousand subjects will be chose n randomly by the Amer: Association of Critical Care Nurses (AACN), from the membership of AAC: and will be asked to complete, via mail, a 25-item test (Clinical Simulation: Pulmonary Artery Pressure Measurement) and demographic dat sheet. A reminder card will be mailed one week after the initial mail: The instrument will be coded to demographic data, but not name. Descriptive and correlational data will be collected. The simulation test will be developed and validated prior to data collection.

III. HUMAN SUBJECTS:

	Yes	No
Are any subjects under 18 years of age?		X
Are any subjects confined in a correctional or detention facility?		X
Is pregnancy a prerequisite for serving as a subject?		X
Are fetuses in utero subjects in this research?		X
Are all subjects presumed to be legally competent?	X	

IV. PRINCIPAL INVESTIGATOR: I certify that the information provided above is correct and that, to the best of my ability to judge, this research qualifies for exemption and will be conducted in accord with the general principles stated in the UW handbook, Vol. IV, Part II, Ch. 2, Sect. 1.

PRINCIPAL INVESTIGATOR'S SIGNATURE __Elizabeth J Bridges__ DATE __1 Apr 91__

V. CHAIR, DIRECTOR, OR DEAN: I certify that this research is exempt from Federal regulations and that it is in accord with the general principles stated in the UW handbook, Vol. IV, Part II, Ch. 2, Sect. 1.

SIGNATURE _____ TITLE __Chair__ DATE __5/4/91__

Send completed form to: Grant and Contract Services, Human Subjects Division, AD-22.